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BILATERAL TRIGEMINAL TIC

ITS ASSOCIATION WITH HEREDITY AND DISSEMINATED SCLEROSIS

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IN A recent paper[1] I have suggested the use of the terms trigeminal tic and glossopharyngeal tic for the two forms of paroxysmal neuralgia affecting the distribution of these two nerves. In both the type of pain is identical, sudden violent brief paroxysms, often started by the movements of eating, swallowing, or handling the face, with the frequent presence of highly sensitive areas of skin, such as lip, ala nasi, or eyebrow, light touches on which may fire off the painful attacks, and aptly named by Patrick "trigger zones."

The term trigeminal neuralgia may be used in too loose a sense to include a number of varieties of pain affecting the face, such as postherpetic neuralgia, a painful sequel of trigeminal zoster, which is increasingly frequent after the age of 50; migrainous neuralgia; the atypical neuralgias, including sympathetic neuralgia and certain recurrent supra-orbital neuralgias which are certainly not migrainous; neuralgia due to irritation of the nerves at the base, and especially that rare form of flashing pain which is liable to affect sufferers from medullary sclerosis following thrombosis of the posterior inferior cerebellar artery.

Thus there may be said to be many trigeminal neuralgias, and it seems to me eminently desirable to use a definite term to describe the paroxysmal form, in which no organic signs of damage to the nerve can be found, such as motor weakness or sensory loss, and in which the pain is arrested by nerve blocking, either by operative section, or by alcohol injection.

Furthermore, precisely similar neuralgic attacks, both in character of the painful spasms and in their reaction to treatment, as well as in the absence of any evidence of trigeminal lesion, are met with in a certain proportion of cases of chronic spastic paraplegia, mostly disseminated sclerosis. There appears to be a rare group also in which typical trigeminal tic appears as a sequel to the sudden onset of trigeminal neuritis, at an interval of weeks, months or years after the fading of the trigeminal anesthesia caused by the neuritis. I have met with three such cases only, in my experience.[1] The type of pain in these cases may be identical with that of the usual trigeminal neuralgia, and is completely relieved by alcohol injection of the gasserian ganglion. They are thus distinguished from the postherpetic cases and those secondary to medullary thrombosis.

The terms trigeminal tic and glossopharyngeal tic will, I suggest, give definiteness to a type of neuralgia whose clinical history and symptoms are clear cut and uniform to an unusual degree among diseases, but whose pathology is at present obscure and unproven. That it may be impossible always to separate this type from rare cases, such as irritation by basal tumors, is a necessary but unfortunate admission in our present state of clinical experience.

Indeed, Dandy[2] states: "It is impossible to differentiate by clinical tests those cases in which the tic douloureux is caused by a tumor." Of a total of 250 cases treated in his clinic at Baltimore he found 18 tumors in the posterior fossa as the cause of the neuralgia, and in other cases arterial loops and venous branches crossing the nerve so frequently that he considers that in one-third of his cases of trigeminal neuralgia a gross lesion was responsible for the pain.

This proportion seems to me incredible, in view of the constant relief to pain afforded by gasserian injection. He met with eight bilateral cases, or 3.2 per cent in his series.

Frazier uses different terms, such as major neuralgia and tic douloureux when speaking of the same disease, and it seems to me that in order to avoid misunderstandings, which are easy to slip into among so many forms of neuralgia which affect the face and head, precision of nomenclature is a necessary postulate.

Trigeminal tic and glossopharyngeal tic are therefore, I claim, syndromes and not diseases, but may be caused in various ways, the most common by far being that form with which we are all so familiar, and which I believe to be dependent on some irritative process at the periphery of the nerve endings, acquired from dental, sinus, or tonsillar sepsis. Frazier does not help us to clarify our thoughts when he writes:[3] "We are convinced that in tic douloureux, unassociated with organic disease, there is no organic lesion as the etiologic factor." He goes on to suggest that the pain may be due to vasospasm, claiming the paroxysmal character of the pain in support of this thesis. The sudden violence of the neuralgia in many cases, "like a flash of lightning," is in my opinion entirely opposed to vasospasm as a cause, and is surely proof of its nervous origin. Its character indeed may be compared with the lightning pains of tabes dorsalis.

No unbiased analytical mind can possibly accept the view that a neuralgia which is so anatomic in its distribution, and never crossing the midline, except when both sides are separately affected, can be idiopathic and a function of normal tissue. Still less can it be of psychogenic origin; there must be a pathologic cause for it, and all the clinical facts point to its site at the periphery of the nerve endings.

Disseminated Sclerosis.—With regard to the occurrence of trigeminal tic as a complication of disseminated sclerosis, I drew attention to its frequency in 1926[4] when I had met with 17 examples. Since then many observers have confirmed this, notably H. A. Parker from the Mayo clinic,[5] and several of

my neurologic colleagues in London have also observed the association. Olivecrona[6] refers to it as a symptomatic neuraliga. The neuralgia is in every way characteristic of the usual paroxysmal trigeminal neuralgia, in severity and type of pain, trigger zones, and reaction to treatment by alcohol injection or root section. The only point of difference is the greater proportion of bilateral cases, seven out of 41 of spastic paralysis with tic, or 17 per cent in my series. This is not surprising if my view of the pathology of trigeminal tic be accepted, that it is due to a terminal neuritis of dental nerve filaments, though the patient may have been edentulous for years. If in addition there are sclerotic plaques in the medulla adjacent to or invading the spinal roots of the fifth nerves, bilateral trigeminal tic would be much more likely to occur, as in disseminated sclerosis.

Frazier[3] states that among over 2,000 cases of trigeminal neuralgia he has never met with a case of disseminated sclerosis. This is to me most surprising, and it is his good luck, or possibly misfortune. I should hesitate to suggest that this was due to any omission to examine the general neurologic condition, because that would account for only a proportion of cases whose disability was not too glaring. Many of my cases have been severely or completely paralyzed. In two of them, both men, neither hand nor foot could be moved, and they lay in bed like mummies, unable to move, and everything had to be done for them by nurses. On the other hand some cases will not mention any weakness of legs until specially asked, when it becomes evident that they have been becoming unsteady on their feet, with difficulty in walking, for perhaps a year or more before the pain commenced, and the plantar reflexes will be found to be extensor in type.

Frazier's proportion of bilateral cases is also much less than mine, 1 per cent as compared to 4.5 per cent, and he disregards my figures because he says they include a number of cases of disseminated sclerosis. The facts are that of my 856 private cases, 39 were cases of bilateral tic, and if the 33 disseminated sclerosis cases with three bilateral neuralgias are withdrawn, that leaves 823 cases, 36 being bilateral, or 4.3 per cent. Moreover, I have no possible doubt that were Frazier to examine a case of disseminated sclerosis suffering from the type of trigeminal neuralgia that all my cases have had, he would at once admit the identity of the neuralgia in type with that which he and others describe as major trigeminal neuralgia, being paroxysmal, with no detectable anesthesia, and showing trigger zones, and in having the pain started in the usual way by handling the face, or eating, or other movements. They are, moreover, relieved in exactly the same way, and to the same extent, by the operations of nerve or ganglion injection, or root section.

I have notes of 41 cases of chronic spastic paraplegia complicated by trigeminal tic, 33 of which (18 women and 15 men) were seen in private practice, three being bilateral cases. In addition I have notes of eight cases treated in hospital, four of which were bilateral, all being women.

Although the majority of my records of hospital cases of the usual type

of trigeminal tic have not been preserved, I have notes of about 300, which for one reason or another I considered interesting, and among them are probably all the hospital cases of spastic paralysis with neuralgia that I have seen. Of the total of 41 cases in private and hospital, seven had bilateral trigeminal tic, or 17 per cent, six women and one man. Of my 33 cases in private practice, three were bilateral, or 9 per cent. Very rarely did the neuralgia commence before organic signs of spinal cord disease appeared. In

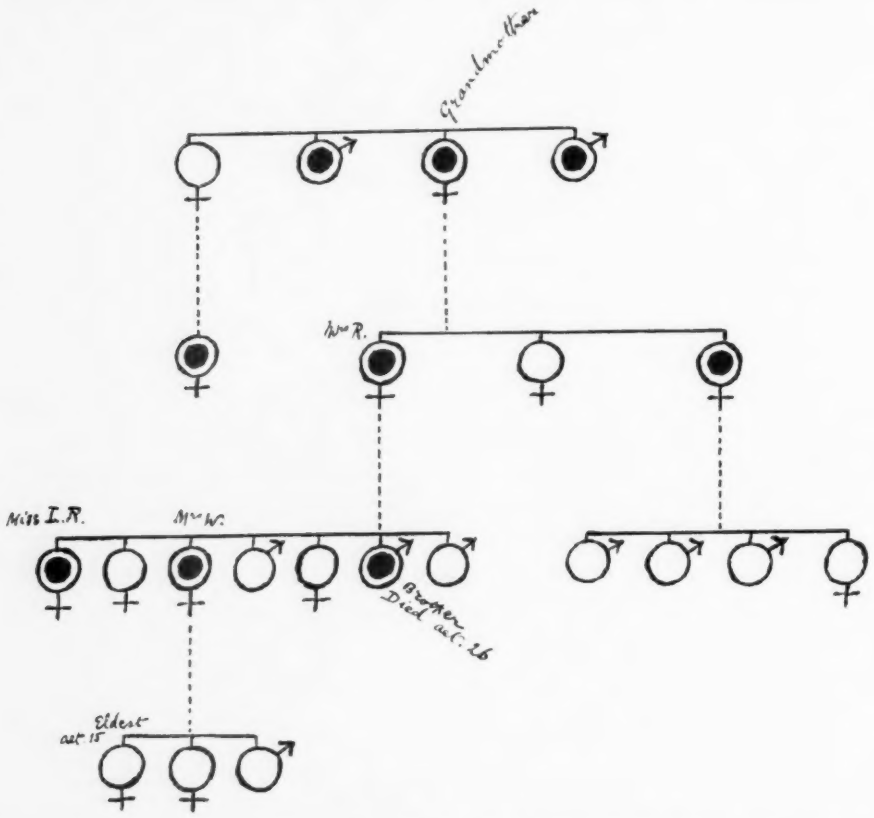

Diagram illustrating heredity of trigeminal tic in the family of my patients, Miss I. R. and Mrs. W. The affected individuals are marked with a black center.

addition to these, in one family the father had trigeminal tic and his daughter developed typical disseminated sclerosis, and in another family I treated the mother for the neuralgia, and her son for disseminated sclerosis. In another I treated the aunt, aged 74, for typical bilateral tic, and her niece for trigeminal tic complicating disseminated sclerosis. The development of the neuralgia in the former lady, aged 74, was interesting. At the age of 44 she had sudden severe pain, beginning in the right upper lip and cheek, coming and going, on and off. Fifteen years later sudden severe neuralgia commenced in the left temple and through the left side of the face, neck and body down to the left foot; similar spasms of pain occurred three times in the same evening,

but never again, except in the left cheek and lower jaw and temple, and half of tongue. The attacks would be started usually by eating, or handling the face, and the pain would occasionally wake her and make her scream. There was a typical trigger zone on the left chin, but the neuralgia on the right side was much less than formerly, mostly prickly pains, like needles, in the right cheek. No injection was considered necessary for the right side, and as she did not want to risk keratitis, in August, 1928, I injected the left second and third divisions separately. This arrested the pain for three years, when I injected her left gasserian ganglion, getting total V.2 and V.3, with analgesia only of V.1, since when there has been no recurrence.

Heredity of Trigeminal Tic.—I have seen in many instances familial heredity of typical trigeminal tic; in one family no less than nine members appeared to have suffered from it, two of them, sisters, being my patients at an interval of 14 years, and both were the subjects of bilateral trigeminal tic. They and their brother all commenced to suffer from the neuralgia between the ages of 16 and 20. He had it only on the right side, and he died at the age of 26 (diagram).

Case Reports

Miss I. R., aged 31, was first seen by me in 1920. Her neuralgia commenced a few months after the age of 16. The first attack of pain was like a flash of lightning in the left lower jaw up to the zygoma. The first and subsequent attacks lasted two to three days, and the crises of pain were precipitated by any movement of the jaw, or sudden noise. In addition she complained of "needle pricks" at the right side of the nose and twitching for several minutes, which were brought on by eating or any facial movements, even occurring spontaneously at times. The pains in the right infra-orbital area alternated with pain in the left lower jaw from the age of 16, though always worse on the left side. She compared the pain to "electric wires."

She was never free of neuralgia for more than a few months at a time, and all her teeth were removed in 1914. She was quite free of all pain between the attacks, the last period continuing for seven months.

February 28, 1920, I injected the left gasserian ganglion by the lateral route, getting temporary anesthesia of V.2 and V.3 for one hour. As the numbness faded, the neuralgia returned, and three days later I again injected through the foramen ovale, getting total trigeminal anesthesia, motor palsy and loss of taste. No further neuralgia has since recurred on the left side. I saw her again in August, 1921, for recurrence of similar neuralgia on the right side, and found the trigeminal anesthesia of the left side was still total except for return of deep sensibility on the eyeball, with partial recovery of the masseter and temporal muscles.

I then injected the right second division of the fifth at the foramen rotundum, getting total right second division anesthesia including pressure, on cheek and upper lip, and on the gum of the upper jaw and palate as far back as the base of the uvula. Two years later the neuralgia attacked the right lower jaw, in August, 1923, becoming severe after a few weeks. In February, 1925, I injected the right gasserian ganglion, getting moderately dense anesthesia of the right second and third divisions. I did not see her again for seven years, when I wrote for her to come and see me. There was still partial anesthesia of the right second and third divisions, and only occasional twitches of pain in the right eye and nose. The left side remained anesthetic as before, but she said she never noticed the numbness there. In August, 1933, the pain returned in the right first and second divisions, and very little anesthesia now remained. I then injected the right

ganglion by the anterior route, getting total trigeminal anesthesia. She was quite free of pain, but unable to bite at all. She wrote that she was not worrying about that. "I am only too glad to be out of pain."

In May, 1935, she wrote, in reply to an inquiry: "My face is still all right. It is quite numb, especially the forehead, nose and lips. The right eye is all right in appearance, but there is always a tingling feeling in it, which makes it not so comfortable as the left side, still I get used to it, and don't mind in the least. I can bite and chew an apple. I was quite a long time before I could eat, and used to get rather downhearted, but after about six months' practice in front of a mirror, I gradually got on and now manage quite well, and I am ever grateful to you for making life worth living for me."

Her long difficulty in learning to eat when bilaterally anesthetic was evidently due to the anesthesia and not to motor weakness.

In giving me the details of her family, she writes: "I know that in all cases the pain has been the same as mine. I had seen my grandmother in terrible agony, although I was only a small child, but I could never forget it."

Her sister, Mrs. W., five years younger, was brought to me in May, 1933. Like her sister, her neuralgia started suddenly with violent pain at the age of 17, in the right cheek and side of nose. This pain recurred frequently, being caused by eating, or handling the face, or by a cold wind. Sometimes the pain shot up through the forehead to the vertex, so severely she might scream aloud.

In 1929 she was injected by another physician, without benefit. Soon afterwards, the neuralgia attacked the left lower jaw, and then the left cheek and nose, 20 years after it began on the right side. She refused to have the ganglion injected, so I injected the right second division only, at the foramen rotundum, getting total anesthesia of cheek, lip and palate on the right side. I hear that she now has pain again, but I do not know which side is troubling her most at present.

I have given the notes of these cases in considerable detail, as they are examples of:

(1) A family with a strong hereditary taint of typical neuralgic tic.

(2) The early commencement of the neuralgia in the two sisters and brother at the age of 16 to 20.

(3) The bilateral affection of the disease in two of these three members of the third generation.

As is also seen in some hereditary mental diseases, the onset of the disorder seems to occur earlier in the descending generation, commencing at 43 in the grandmother, at 32 and 40 in the mother and aunt, and at 16 to 20 in the next generation.

In addition to this family with nine sufferers from the disease in three generations, I have notes of nine other families in which one parent, father or mother, had suffered from similar neuralgia, and in another two instances one grandparent only. In all but one, in which I was able to get approximate dates, the same tendency to anticipation in the younger generation was found. Thus, a lady whom I treated at the age of 62 had begun to suffer at the age of 44. Her father had suffered similarly from the age of 60 until he was 80, when the severe pains left him, and he lived in comparative comfort until the age of 101. This is the only instance I have met with in which the pains disappeared spontaneously without any injection or operative treatment. In

another instance the mother commenced at 68 and the neuralgia lasted until her death at 76, while her daughter, my patient, began to have neuralgia at 41.

In three families I have treated both parent and child by alcohol injection. In 1912 I first injected Mrs. M. who had suffered for 16 years since she was aged 54. She later developed bilateral trigeminal tic, which continued until both gasserian ganglia had been injected. Her second daughter began to suffer in 1913 from neuralgia of R.V.3 at the age of 33, passing off after a few weeks, and recurring severely after 18 months. No treatment arrested this until in 1916 I injected R.V.3 at the foramen ovale. This gave complete relief for over five years, when there was recurrence for a month, then free for 18 months. I then, in 1923, injected the right ganglion, getting nearly total anesthesia, since when there has been no recurrence.

A man who commenced neuralgia between 60 and 70 in R.V.2 I injected five times in seven years at the foramen rotundum. After the last injection in 1924 at the age of 81, he had no recurrence of the pain when still living at the age of 89. His son commenced typical tic at the age of 50, also in R.V.2. He described his pain as originating like little electric shocks in the right cheek, which gradually increased in range and severity, the pain being hot in character, "like forked lightning," and started by movements of eating and talking, or a draught of air. A somewhat unusual character of the pain in his case was that it would often wake him as many as twenty times in a night, making him scream out. Removal of all his teeth had given no benefit. Injection of R.V.2 gave relief for a year in 1921, and in 1923 I injected the right gasserian ganglion, getting total anesthesia of the first division and less complete of the second and third divisions. He had no pain for nine years, until 1932, when pain recurred in the right cheek. He was still totally anesthetic on the first division, the eye and cornea having kept in normal condition, but there was complete return of sensation on V.2 and 3. Reinjection again arrested the pain.

In 1913 I injected the right ganglion in a woman aged 55, who began to suffer in the right upper and lower jaws nine years before, at the age of 46. Total trigeminal anesthesia was produced, and persisted, and seven years later there had been no recurrence. Her son then came to me for similar neuralgia in the left second division which had persisted for four years since he was aged 34. I injected his left gasserian ganglion, getting total anesthesia, which persisted unaltered a week later, and he was quite fit and well. I have had no news of him since.

I have already referred to an instance of a lady aged 74 whom I saw in 1928 with bilateral trigeminal tic, which commenced on the right side at the age of 44, and 15 years later attacked the left side more severely, after which the right side did not trouble her much. Her niece commenced to suffer from left trigeminal tic at the age of 37, which I treated 18 years later. Meanwhile, four years after the neuralgia commenced, her niece began to develop symptoms of disseminated sclerosis commencing with weak legs, and two years later retrobulbar neuritis, with central scotoma, later clumsiness of hands

and sphincter weakness. On examination she had typical disseminated sclerosis, and there could be no possible doubt that her trigeminal tic was indistinguishable from the ordinary variety, and, like them, it was arrested by alcohol injection.

Percentages for Sex and Side Affected.—In studying my statistics of trigeminal tic, two points have constantly struck my notice, the greater frequency of the disease on the right side, and the greater proportion of female sufferers. Of 856 cases seen in private practice, 39 were bilateral, and of the remaining 817 there were 491 right sided to 326 on the left side. Of this total, 550 were women and 306 men. By including 284 hospital cases of which I have notes, my total figures are 1,140 cases, of which 61 per cent were right side and 39 per cent left side. Seven hundred and forty-eight were women and 392 men, or nearly 66 per cent female to 34 per cent male. I have notes of 60 cases of bilateral trigeminal tic, 39 in my private practice, and 21 at my two hospitals, the private cases being equivalent to over 4.5 per cent of my total 856. Of these 60 bilateral cases, the numbers of women were 31 and 17 in the two groups, a total of 48, or 80 per cent, of all the bilateral cases. Frazier's figures, 19 women in 23 bilateral cases, shows the same preponderance in the female. Thus the greater liability of the female to trigeminal tic is, as one would naturally infer, augmented when the disease becomes bilateral, from a two to one to a four to one proportion.

Treatment of Bilateral Trigeminal Tic.—A few of my cases were bilateral from or near the commencement, but in the majority there was an interval of many years between the onset on the two sides, and when both sides were affected, one is usually much worse than the other until the first has been cured by operation or injection. In my experience alcohol injection of the ganglion on each side for bilateral trigeminal tic has been very satisfactory, even though in two of the cases I had to inject both sides at the same sitting, on account of the pain on both sides. It is true that jaw drop then results, and it will be about three months before sufficient biting power is regained. Nutrition can be easily maintained with soft and liquid foods. Owing to the motor root of the trigeminal nerve deriving its nuclear supply from within the pons, the motor fibers regenerate after alcohol injection, even though the sensory nerve cells in the ganglion are completely destroyed and the resultant anesthesia is permanent. If six months or more intervene between injection of the two sides, then rarely is there any real disability in eating, except on account of the bilateral numbness. In some people this may necessitate practice in front of a mirror, as in my hereditary case previously described.

Repeated injection of the foramen ovale may be followed by permanent motor weakness, and if this is done several times bilaterally, it is conceivable that permanent jaw drop might result. I have, however, never yet met with this sequel.

Three years ago I had to deal with an old lady of 88, with severe bilateral neuralgia of the third division, and I injected both foramina ovale on succeeding days. After the second injection she was free of all pain, but com-

plained much of the numbness, as very old women sometimes do, when the pain has vanished. This is, I think, due to failing memory. For some months she was in trouble with jaw drop and had to be fed on minced and soft foods, but later recovered her bite and her pleasure in life, as the following letter recently received from her daughter testifies: "The operation was certainly a very great success, and she has had no return of that terrible pain, which had grown to be almost unbearable. Her mouth has never quite regained its full use, though I think the little disability that remains is largely due to her age and her needing new teeth, which she won't have, because at her age, 91, she is too old, she says." Would a neurologic surgeon have undertaken a bilateral fractional root resection at her age, 88, and with as good a result?

One more bilateral case may be cited as a successful example of ganglion destruction by injection on both sides.

Mrs. M. H. commenced to suffer with neuralgia in the right second and third divisions of the fifth nerve at the age of 36. She used to rub the face to stop the pain, and rubbed away half her right eyebrow. Seven years later, in February, 1913, I injected the right gasserian ganglion, producing permanent total trigeminal anesthesia, unaltered after five years, when the right masseter was found to have recovered completely. Neuralgia commenced in the left second division in July, 1918, with trigger zone on lower lip. The left second division was injected, with total second division anesthesia. She had freedom from pain for 16 months, when all anesthesia on the left side had faded away. I then did a left ganglion injection, obtaining total anesthesia. The left masseter was weak, but no obvious loss of biting power. She then had bilateral total trigeminal anesthesia. Later she described that her food felt like chaff in her mouth, that she could not feel or taste it until she began to swallow. She also mentioned that she could not feel herself kiss her husband. In spite of this additional handicap, she declared the cure of the pain was worth it ten times over.

Character of the Pain.—It is remarkable how often the type of pain is complained of as being hot. Thus it may be compared to "red hot wires" or "fishhooks," "electricity," "like 500 needles shot into the face," or "like a bunch of fireworks exploding." The suddenness of onset and cessation of the pain is characteristic, though after it has passed there is often left behind a sensation of burning in the face for a short time. In the most typical forms all movements such as eating, blowing the nose, cleaning the teeth, washing or rubbing, especially very light touches on the cheek, or a draught of air, will start a painful spasm, but in some of the cases eating has no bad effect, though handling the face may always start an attack during a period of suffering. Some few cases instinctively rub the face to check the pain, and occasionally an eyebrow or mustache may be rubbed away by this constant maneuver, and I have seen the skin of the face on the affected side quite calloused and thickened, so that the two profiles appeared like different people. Sometimes while the pains are recurring regularly there may be apparently hypo-esthesia to pin prick on the cheek and lips, which may lead to a suspicion of an organic lesion, though the sensation will be found to be equal and normal when the bout of attacks has subsided. More commonly, while the painful

attacks are going on, the cheek is more sensitive to pin prick on the affected side.

Sometimes the painful spasms recur at almost regular intervals of three to three and one-half minutes. One such case that was sent to me could not restrain himself from throwing up his hands and holding them or his pillow tightly on to his face as soon as the pain attacked him. An attempt had been made at another hospital to inject him, but had failed for this reason. I asked him if he could give me a second's warning before doing this, which he promised to do, so having got everything ready, and the landmarks drawn on his face and painted over with iodine in spirit, I waited until a paroxysm ceased, and then immediately novocained the skin and deeper parts and quickly sank a 2¾ inch needle by the lateral route to his foramen ovale, and was successful in anesthetising the nerve with novocain, and thus arresting the next spasm before it became due. After that the alcohol injection was proceeded with slowly, as usual. Another case of a young phlegmatic Scot had been sent to me from Burmah, and was having spasms of pain every one to one and one-half minutes for weeks, night and day. He had had practically no sleep and was brought straight from the ship to the nursing home. The spasms of pain continued up to the moment of my finding the foramen ovale with the needle; fortunately he was able to keep his hands and head fairly still, or a general anesthetic would have been necessary, such as morphia-hyoscine, or avertin. Evipal is, I think, less suitable.

Methods of Injection.—In the large majority of cases local novocain is sufficient and far the best, if time is taken and nothing hurried. The patient's cooperation is most valuable in getting the best results, as their replies to repeated tests with a pin are essential for a perfect approach to the hole, to use a golfing phrase. No alcohol should be injected until anesthesia of the chin and lower lip have been obtained with novocain (2 per cent). Then slowly, drop by drop, 90 per cent alcohol is injected, never as much as 0.5 cc. pushed in quickly, or trouble may result. Total ganglion anesthesia often results after the first four or five minims, but although this is most encouraging, we must not be deceived into believing that the operation is concluded, and withdraw the needle. Often far from it. Patience is necessary, and we must wait, perhaps ten minutes, before repeated tests disclose the fact that sensation is returning on forehead and cheek. Such anesthesia is due to ganglion shock, the nerve cells being, so to speak, knocked out temporarily like a boxer by a blow on the chin. Slowly, drop by drop, more alcohol is injected, and if the point of the needle is truly placed, then again total anesthesia is obtained, and the same sequence of events may recur three or four times before the anesthesia persists. Even then it may be found that next day partial sensation has returned. Occasionally permanent and total anesthesia results from the first injection of as little as five or six minims of alcohol (0.3 cc.). Care must, of course, be taken before injecting any alcohol, to see that no C.S.F. drips from the needle, or the alcohol will seep round to the internal auditory meatus, causing intense vertigo and nausea, nystagmus to the opposite side, and perhaps even nerve deafness and facial palsy. This I

usually speak of as the vestibular syndrome. The same result may follow too rapid injection into the ganglion tissue, causing it to burst, with leakage of alcohol along the base.

Immense patience is essential for successful alcohol injection of the ganglion, often one and one-half to two hours being necessary to produce the best result. If done in this manner, I consider it is superior to any operative root section, as all danger to life or to the effects of cerebral compression are avoided. I agree entirely with Härtel[7] that it is not the mild atypical neuralgias that should be injected, but the fierce unbearable trigeminal tics. "Nein und nimmermehr! Nicht die 'Minorfälle' sondern die 'Maximusfälle' gehören der Ganglioninjection."

His figures are instructive. Of 73 German cases he obtained total anesthesia in 47, or 64.5 per cent, and 68.5 per cent remained cured. Of 98 Japanese cases he obtained total anesthesia in 62 per cent and cure in 81 per cent, with return of pain in only 19 per cent. His statistics agree approximately with mine in showing a relative high proportion of right sided cases, 63 per cent to 37 per cent on the left side. He had, however, more male than female cases, out of a total of 213, which differs strikingly from my experience.

Motor paralysis usually passes off gradually after a lapse of three months, and bilateral ganglion destruction by alcohol injection is compatible with good power of eating and chewing, as I have proved many times. Both sides may even be injected at the same sitting in the emergency of severe bilateral neuralgia, for temporary jaw drop of a few months is not to be compared with the agony of persistent painful spasms.

Injection of Inner Two-Thirds of Ganglion.—In many cases of violent neuralgia affecting the first and second divisions only, I have been able to leave good sensation in the lower jaw and tongue, and yet produce total and permanent anesthesia of the upper portion of the face. This I do by pushing my needle one-quarter inch farther through the foramen ovale before injecting any alcohol, and after anesthetizing the third division with novocain. The preservation of sensation on the lower jaw and tongue is valuable in such patients for purposes of eating and enjoying their food, as if these parts are anesthetic they invariably choose to eat on the other side, just as they do when suffering from neuralgia.

Formerly I invariably used the lateral route to reach the foramen ovale, between the zygoma and the sigmoid notch, but during the last three years I have used mainly the anterior route which Härtel described, when I wish to inject the ganglion. Owing to its steeper approach, there is greater ease in many cases in passing the needle between the lips of the foramen, but a four inch needle is required instead of one of two and three-quarter inches. If the route is first well prepared with novocain with a fine needle, I do not find much greater risk of hematoma than by the lateral route, and it is always easily controlled.

Sometimes I fail to get good ganglion anesthesia by the anterior route, and then may succeed perfectly by the lateral route, and it is certainly very useful to have an alternative approach. This greatly increases the proportion

of cases in which dense and permanent ganglion anesthesia is obtainable, so that very rarely should failure result. Even an open operation by a neurologic surgeon sometimes fails to section the nerve as desired.

Frazier has greatly advanced the technic of the open surgical operation by his perfection of the fractional root section method and saving the motor root, and it would appear from his statements that he does not admit recurrences of neuralgia. By Dandy's cerebellar route a useful alternative approach is afforded, which probably offers greater security for the motor root. Dandy, however, in his subtotal series, confesses to four recurrences, and judging from Hutchinson's and other partial gasserectomies, in which recurrence of the neuralgia is not uncommon, I feel convinced that to ensure absolute safety of nonrecurrence a total root section or ganglion destruction is necessary. In the small proportion of cases in which the ophthalmic branch is definitely involved together with the second division, it would be most unsafe to leave the inner portion of the root undivided, in order to minimize the risk of keratitis, for a second gasserian operation is a very serious risk. On the other hand, injection of the inner portion of the ganglion, leaving the third division sensitive, is a negligible risk in case neuralgia spreads into the lower jaw or tongue, for the injection can be easily repeated, and the third division and outer portion of the ganglion dealt with as if it were a fresh case.

All gasserian operations are difficult, and require highly skilled neurosurgery, and should never be trusted to a surgeon who has not studied and practiced the operation carefully. Much the same may be said of alcohol injection. Long practice and familiarity with the anatomy and the methods of injection and its difficulties are necessary to produce the best results, and should, therefore, be left to the hands of experts, and here again I may quote Härtel[8]: "Nicht nur die Ganglionoperation, auch die Ganglioninjection gehört in Meisterhände." Unfortunately, many surgeons attempt it without due study, owing to its comparative safety, and consequently fail, and even sometimes make tragic blunders. Thus alcohol injection is often looked upon askance and rejected by physicians and surgeons as at best only a temporary palliative, but it is, when properly performed, one of the most delicate and beautiful operations in minor surgery, equally capable with the open operation in effecting a permanent cure, and with no risk to life.

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PENETRATING WOUNDS OF THE BRAIN

AN EXPERIMENTAL STUDY

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THE extremely variable tolerance of the brain for foreign bodies introduced through penetrating wounds of the skull has led to a wide diversity of opinion regarding their treatment. Many cases of prolonged retention of foreign bodies without symptoms are on record. Elliot's[110] patient was known to have had a bullet in his brain for 65 years without symptoms, and other cases have survived 30 years (Martin[121]), 27 years (Tanaka[133]), 18 years (McLean[204]), piece of steel; Schlöss,[175] knife blade, 17 years; (McEachern[203]), surgical probe; and similar periods.

On the other hand, latent brain abscesses have made their appearance after 16 years (Urechia[165]), 11 years (Bregman[155]), 10 years (Ransohoff[164]), and lesser intervals; and innumerable cases of late epilepsy are reported.

By far the most common foreign bodies lodged in the brain have been bullets and the voluminous literature of the various periods of war has abounded in reports and discussions on the subject. However, in less frequent instances the brain has been penetrated by almost every other conceivable instrument. Of these, knife blades are the most common (Babcock,[167] Blaine,[168] Brown and Birch,[169] Dretzka,[170] Guleke,[171] Karschulin,[172] Pepper,[173] Schloffer,[174] Schlöss,[175] Selwyn,[176] Slee,[177] Sommer,[178] Wilkins[179]) and cases have been reported of penetration of the brain by pitchfork prongs (Kesteven,[198] Hooper[195]), a crochet hook (Franklin[191]), a knitting needle (Annandale[181]), breech-pins (Cunningham,[185] Thompson,[211] Kemper,[196] Burr,[184] Noyes[205]), surgical instruments (McLean,[204] McEachern[203]), pieces of wood (Pridioux,[207] Matthews,[202] Ferguson,[189] Key and McCrummen[199]), the rib of an umbrella (Donkin[186]), a crow-bar (Harlow[194, 183]), iron rods (Swanson,[209] Pugh[208]), nails (Levin,[200] Felty[188]), and other bizarre objects.

In the first half of the last century, most surgeons adhered to the conservative advice of Sir Benjamin Brodie[14] (1828), who stated that no foreign body should be removed unless it lay on the surface of the brain in an already open wound. By the middle of the century, some surgeons had begun to remove foreign bodies in some instances. In the "Medical and Surgical History of the War of the Rebellion"[86] are reported 85 cases of bullet removal, of which 43 are listed as "recoveries." In 1868, Andrews[3] reviewed 73 cases of retained bullet and advocated removal ("and the sooner the better") of all superficial foreign bodies which could be definitely located and easily approached. Ten years later, the same opinion was expressed by Wharton[88] who collected 316 cases. Of these, the foreign body was removed in 106 cases

with 72 recoveries and not removed in 210 cases with only 88 recoveries. Similar opinions were expressed by Morris[55] (1887), and Papaillon[61] (1894). Hewett and Lidell[40] stated (1881): "Every reasonable effort should be made to extract foreign bodies when lodged in the brain."

With the discovery of roentgen rays (first employed for the location of cranial and intracranial foreign bodies in 1896 by Stubenbord[212] of New York, Eulenberg[213] and Schier[214] of Berlin, and Fowler[215] of Brooklyn, in the order named), the removal of many more foreign bodies was attempted. Indeed, Phelps[126] wrote, "There is probably no authenticated case . . . in which the bullet left in the brain substance has failed to work mischief," and in 1910 Da Costa[21] stated, "Practically every lodged bullet (in the brain) constitutes a fatal condition and it should be removed if possible, even if there are no symptoms."

With the beginning of the World War, another controversy further complicated the situation. Prior to this time, the commonly accepted method of treatment of penetrating wounds of the brain had consisted in removal of fractured bone, leaving the wound, including the dura mater, open, allowing a cerebral hernia to develop and the wound to heal (if it would) by secondary intention (von Bergmann,[11] Papaillon,[61] Doyot[26]). This method was advocated again during the war (Whitaker,[89] Moulonguet and Legraine,[56] Hunt[45]), but was condemned by Bárány,[8, 9] Cushing,[18, 19, 20] Willems[90] and many others, who advocated early operation, complete débridement and primary suture.

The diversity of opinion continued throughout the war, both as regards foreign bodies and the open and closed methods of treatment. Gross and Houdard[34] advocated primary suture of wounds without removal of foreign bodies. Sargent and Holmes[68, 69] stated that only readily accessible foreign bodies should be removed. On the other hand Dretzka,[25] Tilmann,[77] Mathieu,[53] Anderson[1] and others advised primary suture after removal of all foreign bodies. Moulonguet and Legraine[56] thought all foreign bodies should be removed and the wound left open. In 1918, Cushing[19, 20] advanced the rational doctrine that foreign bodies should be removed whenever the damage of removal did not exceed the trauma of the original penetration. He devised the catheter-suction method of débridement and employed the "tripod" or "Isle-of-Man" incisions with primary suture in all cases. These principles were widely accepted (Horrax,[42] Neuhoff,[59] Harvey,[38] Willems[90]).

Many special methods came to be employed. Localizing and extraction apparatus was designed by Hirtz,[246] Ferguson,[245] Banzet,[244] and Swanberg,[248] electromagnets were employed by Lee,[238] Cords,[234] Lefort,[239] Cushing,[235] Sargent and Holmes,[242] La Peyre,[237] Mauclaire,[240] Spick and Jauréginberry,[243] Kümmell[236] and Regard,[241] and extraction under the fluoroscopic screen was advocated by Cazamian,[227] La Peyre,[229] Rouvillois,[233] Lee[230] and others.*

* In 1903, Holzknecht[221] anticipated the fluoroscope by making plate exposures at operation to determine the location of a previously inserted probe in relation to the foreign body.

De Martel[52] was the first to advocate the use of local anesthesia, which was widely employed thereafter.

Concerning the late treatment of retained foreign bodies, it was generally agreed that only those which were very superficial or those which were causing symptoms should be removed (Bagley,[92] Coleman,[103] Frazier and Ingham,[112] Demmer,[106] von Eiselsberg[109]). This was finally expressed as the official opinion of the Interallied Surgical Conference.[116]

That the diversity of opinion alluded to above exists is ample evidence that the factors underlying the tolerance of the brain for foreign bodies are poorly understood. On only one of these factors is there unanimity of opinion: all observers agree that penetration of the cerebral ventricles by a foreign body greatly increases the danger of fatal infection (Harvey,[142] Cushing,[138] Trotter and Wagstaffe,[152] Regard,[150, 151] Horrax[144] and others). Other factors, such as the duration of retention, and communication with the subarachnoid space or surface of the skin, have been generally ignored or disagreed upon.

Experimental work on the subject has been meager. In 1862, Flourens[250] placed "leaden bullets" upon the surface of the brains of rabbits and dogs and observed that the presence of a foreign body seemed to predispose to "inflammatory action." In 1869, Philipeaux and Vulpian[254] produced wounds in the left hemispheres of three dogs and observed that two died, while one recovered. Kramer and Horsley[251] studied the immediate effects of gunshot wounds on the circulation and respiration. Hortega and Penfield,[255] and Penfield and Buckley,[252] have made careful studies of the histology of experimental cerebral wounds.

The present report is concerned with a study of some of the various factors influencing the outcome of penetrating wounds of the brain.

Methods.—Dogs weighing from five to eight kilograms were used in all experiments. Experiments were of two general types: those in which a short, sharp nail about two millimeters in diameter was inserted through the skull to varying depths and left in place for varying lengths of time and those in which a lead air rifle shot (about two millimeters in diameter) was introduced into the brain through a small operative opening in the skull. In the former series, the animals were given morphia 0.13 Gm. (1½ gr.) one-half hour previously; in the latter they were anesthetized with ether.

The nails used were filed until very sharp and, with the animal's head firmly held on a wooden block, surprisingly little force was required to pierce the skull to any desired depth. Once through the skull, the nails were held firmly by the bone and could not be withdrawn without the use of a claw hammer or other instrument. After a period of two to four days, pressure necrosis (and, in some instances, infection) of the bone had occurred to a sufficient extent to render the nails loose and, in several instances, they came out spontaneously. In no instance was a fracture produced. Subsequent to the original procedure, all animals were observed frequently. In several

instances in which obviously fulminating infections developed and at the end of varying periods in surviving animals, they were painlessly sacrificed.

The same site was chosen in all animals for placing the nail or making the opening through which the shot was to be inserted. This was a point approximately 2 cm. above the posterior point of attachment of the left ear to the scalp. This point corresponds to a "silent" area of the brain and in no instance were any abnormal neurologic signs demonstrable immediately after the insertion of the foreign body.

Clinical neurologic signs are extremely difficult to evaluate in the dog. Only those which were quite definite will be mentioned in this report.

The nails and shot were not sterilized nor were they deliberately contaminated with any particular organism. Detailed bacteriologic studies were not made, but, in several instances in which cultures were made from widespread cerebral infections mixed growths of staphylococci, streptococci and *Bacillus coli* were obtained.

RESULTS.—*Group I.*—In 12 dogs, nails were inserted to a depth sufficiently great to penetrate the lateral ventricle and allowed to remain in place throughout the animals' lives. Approximately 1 cm. of the nail was left protruding from the scalp. The results are given in Table I.

TABLE I

The Effect of a Foreign Body Penetrating and Left Indefinitely in Communication with the Skin Surface

Dog No.	Duration of Life in Days	Ventricle Penetrated	Blood in Frontal Sinus	Postmortem Findings
N 1	2	Yes	Yes	Left ventricle distended with pus. Extensive left sided meningitis
N 2	2	Yes	Yes	Left ventricle distended with pus. Extensive left sided meningitis
N 3	2	Yes	Yes	Left ventricle distended with pus. Extensive left sided meningitis
N 4	1	Yes	No	Large hemorrhage in left ventricle. Clot in posterior fossa
N 5	2	Yes	Yes	Both lateral ventricles full of pus. Purulent meningitis on left
N 6	2	Yes	No	Pus-filled nail tract and ventricle on left. Left sided meningitis
N 7	14*	Yes	No	(1) Extensive osteomyelitis. (2) Dura adherent over left hemisphere. (3) Puncture wound sealed off. (4) Ventricular system distended pus
N 8	26†	Yes	No	Chronic healing meningitis; chronic inflammation and necrosis extending to ventricle and involving choroid plexus. Multiple microscopic abscesses in adjacent area

TABLE I—*Continued*

N 9	6	Yes	Yes	Ventricular system full of pus. Widespread meningitis
N 10	3	No	No	Large left sided subdural abscess, extending to floor of middle fossa
N 11	3	Yes	No	Dura adherent at puncture site. Extensive ependymitis in lateral ventricles; gross pus in third and fourth ventricles and about brain stem
N 12	2	Yes	No	Adhesions over left hemisphere. Pus in left lateral, third and fourth ventricles and about the base; ependymitis in right lateral ventricle

* Nail came out spontaneously on sixth day. Animal died on fourteenth day.
† Nail came out spontaneously on fifth day. Animal sacrificed on twenty-sixth day.

One dog (N 4) died on the day on which the nail was inserted and was found to have a large intraventricular hemorrhage. Six dogs died on the second day, two on the third day and one on the sixth day after insertion of the nail. In eight of these nine animals, the nails were found to have penetrated the ventricles, and a widespread ventricular, cerebral, and meningeal infection was present. In six of these animals, the ventricles were distended with pus. In the ninth animal (N 10) who died on the third day, the nail had not penetrated the ventricle and the necropsy revealed a large subdural abscess.

Clinically, the signs in these nine animals were those of overwhelming infection and general impairment of the function of the central nervous system. Lethargy, rapidly progressing to stupor, coma and death, was present in all. Hemiplegia was apparently complete in Dog N 10 shortly before death. None of the animals had definite choking of the optic disks.

In one dog (N 7), the nail came out spontaneously on the sixth day. The animal ate well, was quite active and there were no apparent symptoms until the twelfth day when he became drowsy, refused food and grew rapidly worse. He died on the fourteenth day. During these two days, he developed high choking of the disks. At necropsy, the ventricular system was full of pus, but the puncture wound in the cortex was sealed off and apparently the outlet from the fourth ventricle was also obstructed, for no inflammation was found in the meninges.

In the single remaining experiment in this group (N 8), the animal was very ill for five days. He was apathetic, staggered and tended to fall to the right. The optic disks became hyperemic and their edges blurred. Pus exuded around the nail. On the fifth day, the nail came out spontaneously, and following this the dog slowly improved until, on the twenty-first day, he was entirely free of symptoms, ate well, and was quite active. The scalp wound had gradually ceased to discharge and was healed. The dog was sacrificed on the twenty-sixth day. Necropsy showed dense adhesions of the thickened dura and arachnoid and a walled off, chronically inflamed tract extending into the ventricle. Microscopically, the choroid plexus was found bound up in the chronic inflammatory process and there were numerous small adjacent abscesses, several of them containing fragments of bone. Compound granular corpuscles filled with phagocytized material were numerous and an area of gliosis extended well into the surrounding cerebral tissue.

In five of these dogs, there was terminal bleeding from the nose and at necropsy the frontal sinuses were found filled with blood. The cause of this is not clear and it is mentioned here as an incidental finding.

Comment.—It was apparent from these experiments that a number of factors might play a part in the development, severity and extent of the infections produced. Among these were the duration of retention of the foreign body, depth of penetration, and communication with skin, subarachnoid space and ventricle. Subsequent groups of experiments were designed to demonstrate the importance of these factors.

Group II.—In 12 dogs, nails were inserted as in Group I, but were removed at the end of 24 hours in four dogs, 12 hours in four dogs and immediately after insertion in the remaining four dogs. The results are given in Table II. In three of the four dogs retaining the nail for 24 hours, death occurred in four, two and three days, respectively. In two of these, the ventricle was penetrated, but in one of them (N 15), the infection found at necropsy did not involve the ventricle.

TABLE II

The Effect of a Deeply Penetrating Foreign Body Left in Communication with the Skin Surface for Twenty-four Hours or Less

Dog No.	Interval Before Removal of Nail, in Hours	Interval Before Death or Sacrifice, in Days	Spontaneous Death or Sacrifice	Ventricle Penetrated	Postmortem Findings
N 13	24	57	Sac.	No	Brain healed. Small cortical scar
N 14	24	4	Sp.d.	No	Small focus of cortical necrosis. No other cause of death found
N 15	24	2	Sp.d.	Yes	Large subdural abscess on left with inflammation in outer portion of nail tract
N 16	24	3	Sp.d.	Yes	Large cortical abscess with involvement of left ventricle
N 25	12	62	Sac.	Yes	Brain healed. Small cortical scar adherent to dura with tiny core extending to ventricle
N 26	12	62	Sac.	No	Brain healed. Superficial cortical scar
N 27	12	3	Sp.d.	Yes	Widespread purulent meningitis, and encephalitis. Left ventricle full of pus
N 28	12	81	Sac.	Yes	Brain healed. Scar tissue core extending to ventricle
N 17	0*	60	Sac.	Yes	Brain healed. Scar tissue core extending to ventricle
N 18	0*	102	Sac.	Yes	Brain healed. Scar tissue core extending to ventricle
N 19	0*	86	Sac.	No	Brain healed. Small cortical scar
N 20	0*	20	Sp.d.	Yes	Died of distemper. Brain healed

* In these animals, the nail was withdrawn immediately after its insertion.

In dog N 14, death occurred on the fourth day, but the necropsy revealed only a small focus of superficial cortical necrosis and the ventricle had not been penetrated.

Dog N 13 recovered and was sacrificed on the fifty-seventh day. The brain was healed and the ventricle had not been penetrated.

Three of the four dogs in whom the nails were left in place for only 12 hours recovered, and, when sacrificed after long intervals, showed no active intracranial infection. In two of the three, the ventricles had been penetrated. The remaining animal (N 27) died on the third day of a fulminating infection involving the ventricle.

All of the four dogs from whose brains the nails were removed immediately after insertion recovered without symptoms at any time and their brains were healed at necropsy. One of them died on the twentieth day of acute distemper. The ventricles had been penetrated in three of the four dogs.

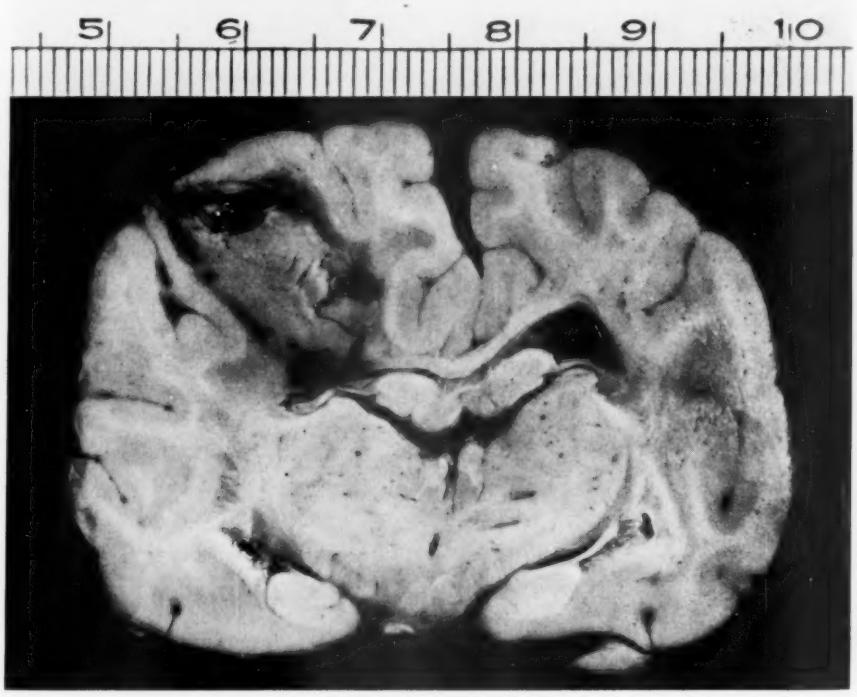


FIG. 1.—Dog N 22. Well walled off abscess 21 days after penetration by foreign body.

Comment.—The experiments in this group showed clearly that the duration of retention of a foreign body communicating with the surface of the skin was a factor of prime importance and that, under these experimental conditions, the period of retention following which recovery was likely was not greater than 12 hours. Sargent[67] has stated that a delay of two to four days after receipt of the wound is advisable before operation is undertaken. The same opinion was concurred in by Cushing[18] in 1916, but he later apparently believed in earlier operation.[19, 20] Trotter and Wagstaffe[80] advocated extraction of foreign bodies within 12 hours if possible. The present experiments are in support of this opinion.

Group III.—In eight experiments, the foreign bodies were inserted as in the preceding groups except that the nails were shorter and the approximate depth of cortical

penetration was 6 to 10 Mm. in four dogs and 2 to 3 Mm. in four dogs. Table III shows the results. In seven of the experiments, the nail came out spontaneously in from two to eight days. Six of the eight animals recovered spontaneously and showed no active infection when sacrificed. One (N 22) died on the twenty-first day and necropsy showed a large, well localized cortical abscess (Fig. 1). The remaining dog (N 32) died of a fulminating infection on the second day. The infection involved the ventricle and it is possible that the latter was penetrated by the nail.

TABLE III

The Effect of a Superficially Penetrating Foreign Body Left in Communication with the Surface of the Skin. Ventricle Not Penetrated

Dog No.	Interval Before Spontaneous Loss of Nail, in Days	Interval Before Death or Sacrifice, in Days	Spontaneous Death or Sacrifice	Approximate Depth of Cortical Penetration, in Mm.	Postmortem Findings
N 21	4	44	Sac.	6–10	Adherent dura. Small cortical scar
N 22	8	21	Sp.d.	6–10	Large, localized cortical abscess. Local osteomyelitis
N 23	4	44	Sac.	6–10	Surface of brain red. Light dural adhesions. Cortical scar
N 24	5	44	Sac.	6–10	Small cortical scar
N 29	3	66	Sac.	2–3	No gross lesion in brain
N 30	4	66	Sac.	2–3	Tiny cortical scar
N 31	2	66	Sac.	2–3	Local dural adhesion to cortical scar
N 32	2	2	Sp.d.	2–3 (?)	Large subdural and cerebral abscess extending into left ventricle

Comment.—It is to be noted that the foreign bodies were retained in all the experiments in this group well beyond the period of safety found in animals whose ventricles had been penetrated (Groups I and II). It is apparent, therefore, that penetration of the ventricle greatly increases the danger of a fatal outcome in such wounds. This is in accord with the clinical opinions already cited.

Group IV.—In eight experiments, the scalp was closed over the nail. In four of them, the head of the nail was left outside the temporal muscle and just beneath the scalp (Table IV). Three of these dogs died of extensive purulent meningitis with cortical infection in three, six and five days, respectively. One of these (N 32) had a large cortical abscess (Fig. 2). In all these, there was purulent exudate and necrosis in the overlying muscle. The fourth dog recovered and at necropsy on the thirty-second day only a small subdural hematoma without meningeal or cortical infection was found. The muscle was densely adherent and fibrosed but was healed.

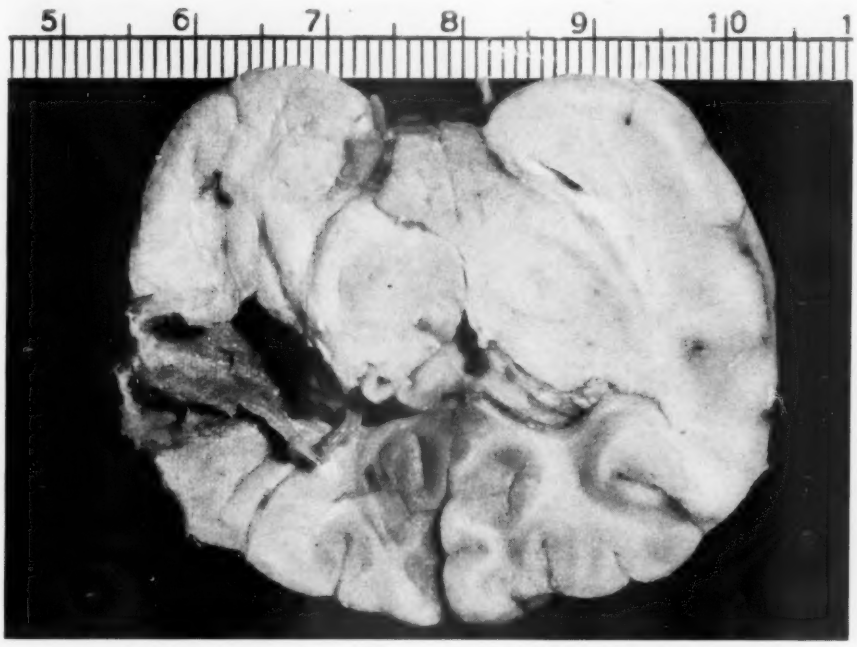


FIG. 2.—Dog N 36. Large necrosing cortical abscess five days after penetration by foreign body.

TABLE IV

The Effect of a Penetrating Foreign Body Over Which the Scalp Has Been Closed

Dog No.	Interval Before Death or Sacrifice, in Days	Spontaneous Death or Sacrifice	Postmortem Findings	Length and Depth of Nail
N 33	3	Sp.d.	Extensive purulent meningitis. Small, shallow cortical abscess	Nail, 1.5 cm long, with head inserted to outer surface of skull
N 34	32	Sac.	Chronic subdural hematoma. No apparent infection about nail	
N 35	6	Sp.d.	Purulent meningitis. Small localized cortical abscess	
N 36	5	Sp.d.	Purulent meningitis. Large localized cortical abscess	
N 37	2	Sp.d.	Small cortical abscess. No gross meningitis	Headless nail inserted through bone
N 38	37	Sp.d.	Well walled off subdural and cortical abscesses	
N 39	74	Sac.	Brain firmly healed about nail. Tip in ventricle	
N 40	74	Sac.	Brain firmly healed about nail	

In the other four dogs, the nails were headless and were inserted completely through the skulls by means of a steel pin with which they were driven. Dog N 37 died on the second day. Necropsy revealed only a small cortical abscess. Dog N 38 died on the thirty-seventh day with a large, well walled off subdural and cortical abscess (Fig. 3A). The other two dogs recovered completely. There was no infection about the nails, although in one of them (N 39) the tip of the nail had penetrated the ventricle (Fig. 3B). The muscle was completely healed in all except N 37. In the three animals in this group who recovered, the nails were completely isolated from the free subarachnoid space by dense dural adhesions.

Comment.—These experiments suggest that communication of a penetrating foreign body with the subarachnoid space considerably increases the danger of fatal infection and that this danger is further augmented if the foreign

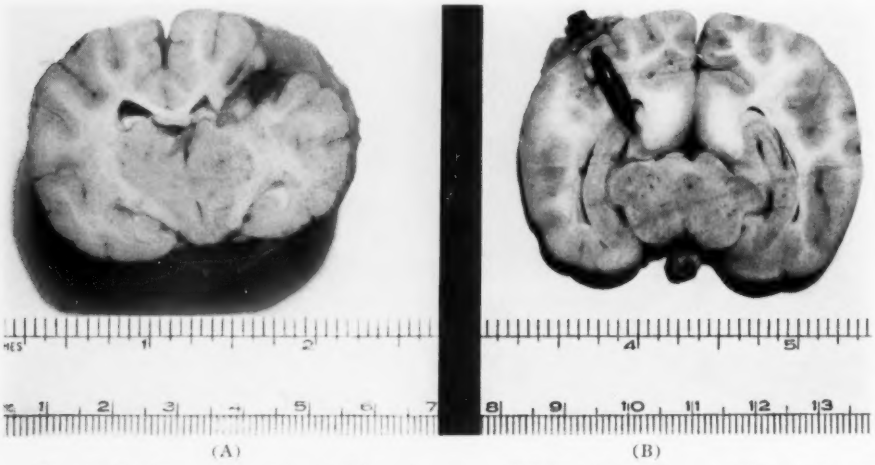


FIG. 3.—Headless nails were inserted into the cortex. (A) Dog N 38.—Large subdural and small cortical abscess, thirty-seventh day. (B) Dog N 39.—The brain is healed about the nail. The tip of the nail has penetrated the ventricle, seventy-fourth day.

body protrudes into the soft tissues overlying the skull. Angerer,[4] in 1914, stated that all patients with wounds penetrating the dura died. This statement is somewhat extravagant, but the danger of communication of foreign bodies with the subarachnoid space has not been generally recognized.

Group V.—In each of eight dogs, an air rifle shot was inserted into the brain through an operative opening in the skull. The operations were performed under aseptic conditions except that no attempt was made to sterilize the shot which were picked up with a fine sterile forceps and inserted into the cortex to the desired depth. The wounds were closed in layers with silk.

In four experiments the shot were inserted to a depth of approximately 10 to 15 Mm. (Table V). All four animals recovered without having developed symptoms at any time. At necropsy, the shot were found firmly bound about with dense fibrous adhesions from which a scar tissue "core" as described by Penfield and Buckley[232] led to the cortical surface (Fig. 4). The ventricle had not been entered in any case.

In the other four experiments, the shot were inserted to a depth of 20 to 30 Mm. Dogs N 52 and N 54 were sacrificed when severe symptoms appeared on the sixth and twentieth days, respectively. In both, the shot had traversed the ventricle and lay deep in the base of the brain. The inflammatory reactions in both were of a chronic nature with organization and fibrosis (Fig. 5). Both showed a low grade basilar meningitis.

TABLE V

The Effect of the Penetration of a Non-protruding Foreign Body Into the Brain

Dog No.	Depth to Which Shot Was Inserted, in Mm.	Period Before Sacrifice, in Days	Postmortem Findings
N 41	10–15	106	Shot embedded in scar with core leading to cortex
N 42	10–15	85	Shot embedded in scar with core leading to cortex
N 43	10–15	254 plus	Still living
N 44	10–15	128	Shot embedded in brain. Thin core leading to cortex
N 51	20–30	50	Scar tissue core marks tract of shot
N 52	20–30	6*	Hemorrhagic, organizing abscess. Mild basilar meningitis
N 53	20–30	92 plus	Still living
N 54	20–30	20*	Small, superficial cortical abscess; tract about shot healing; fibrinopurulent exudate at base

* These animals were sacrificed when severe symptoms appeared.

Dogs N 51 and N 53 recovered completely. In the former, the shot was found at the bottom of a healed tract, as in the previous group. Dogs N 43 and N 53 have been preserved alive for histologic study of a more long standing healing process.

Comment.—The uniform recoveries in the first four experiments of this group constitute strong evidence that the high incidence of fatal infection found in the preceding groups was the result of continuous communication of the foreign body with the skin surface, the ventricle or the subarachnoid space. This is substantiated by the development of infections (though less severe than in previous groups) in two of the four animals in which the ventricle was traversed by the shot. This does not necessarily imply a greater intrinsic resistance to infection on the part of the cerebral tissue, but rather suggests that the greater danger of contamination of the ventricles or subarachnoid space arises from the rapid spread of the infection by means of the cerebrospinal fluid. This is borne out by the findings in dog N 7 (Group I) who survived 14 days with an extensive intraventricular infection which was sealed off from the adjacent cerebrospinal fluid spaces.

Group VI.—In six dogs, nails were inserted into the brain to a depth of approximately 10 Mm. (as in Group III). After varying intervals, under ether anesthesia and with aseptic precautions, the nails and surrounding area of bone (about 2 cm. in diameter) were removed and the dura widely opened. The wounds were then packed open. The results are given in Table VI.

In the first two dogs, the operation was performed two days and in the third three days after insertion of the nail. Active infection was found in two of these three animals. All three made complete recoveries after operation. The wounds drained freely at first, then gradually filled with granulations and healed. N 45 and N 46 were completely healed on the fifteenth day and N 47 on the nineteenth day after operation.

In the remaining three animals, operation was performed five days after insertion of the nail in N 48 and seven days in N 49 and N 50. Dog N 48 died the day follow-

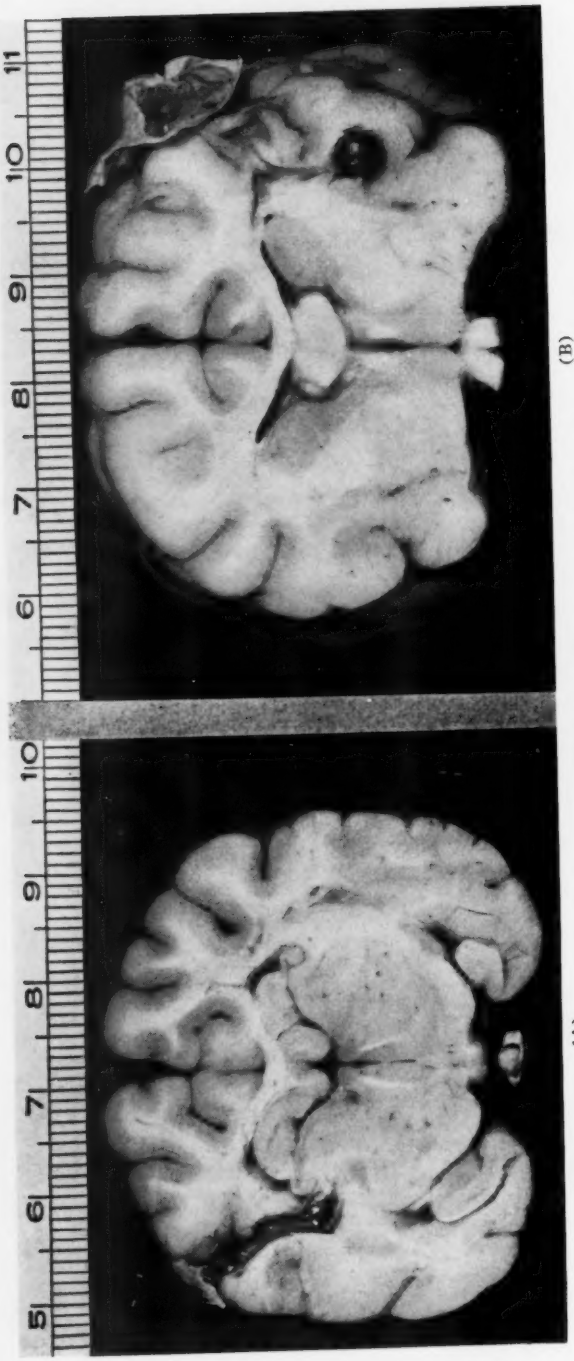
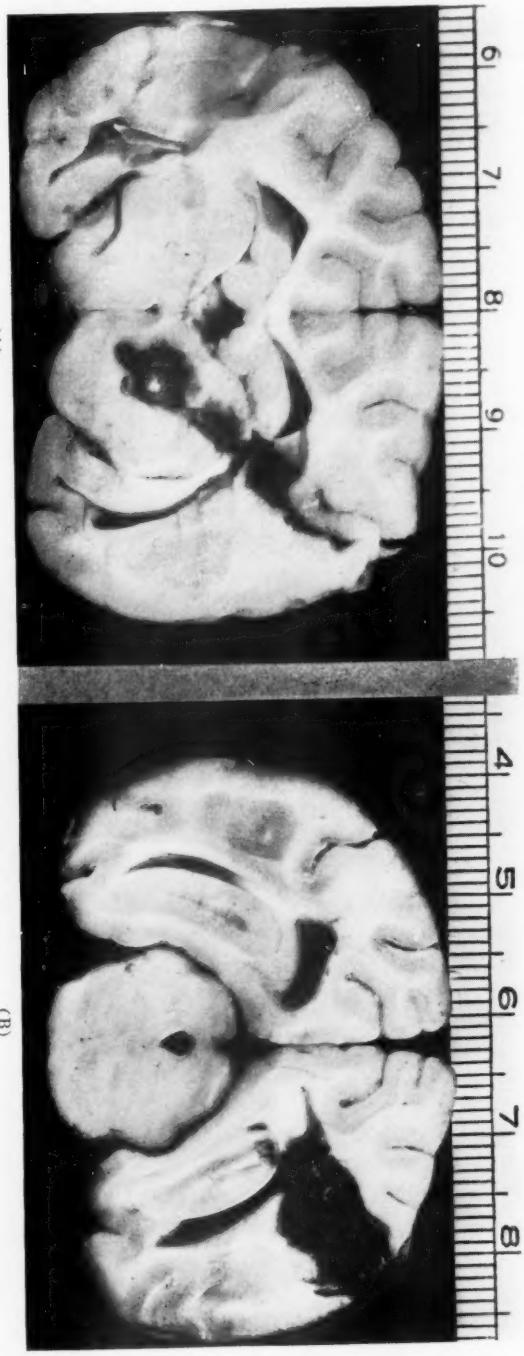


FIG. 4.—Embedded shot with healed "cores." The ventricles have not been penetrated. (A) Dog N 42, eighty-fifth day. (B) Dog N 41, one hundred and sixth day.



(A) (B)

FIG. 5.—Dog N 52. Sections at different levels. (A)—The shot embedded at the bottom of its deep tract. (B)—There is no free pus in the hemorrhagic cavity, sixth day.

TABLE VI

The Effect of the Operative Removal of a Penetrating Protruding Foreign Body with Its Surrounding Area of Bone and Dura Mater. Wound Left Open

Dog No.	Interval Between Insertion of Nail and Operation, in Days	Operative Findings	Postoperative Period, in Days	Spontaneous Death or Sacrifice	Postmortem Findings
N 45	2	No gross infection	84	Sac.	Brain healed. Adhesions. Cortical scar
N 46	2	Small amount of subdural pus	169 plus	—	Still living
N 47	3	Small amount of subdural pus	65	Sac.	Small cortical scar. Dural adhesions
N 48	5	Small amount of subdural pus	1	Sp.d.	Localized cerebral abscess
N 49	7	Cortical granulations. No pus	14	Sp.d.	Walled off subdural abscess
N 50	7	Extensive purulent meningitis	0*	Sp.d.	Extensive meningitis and encephalitis extending into ventricle

* Animal died as anesthetic was begun.

ing operation and was found to have a large localized cerebral abscess. Dog N 50 died as the anesthetic was begun. Hasty removal of the nail with release of pus under great tension failed to resuscitate him. At operation dog N 49 was found to have no actual purulent exudate, but definite evidence of infection. The wound drained profusely and the animal was free of symptoms until the twelfth day after operation. On this day, he was listless and drowsy, refused food and water and looked ill. All extremities were moved well and the reflexes were normal. The following day, high choking of the optic disks was observed. Drainage had greatly diminished. On the fourteenth day the animal died. Necropsy revealed that the infection had become walled off to form a large subdural abscess.

Comment.—These experiments demonstrate that the increase in likelihood of recovery resulting from removal of a protruding foreign body may be augmented by ensuring adequate drainage, providing subdural or cortical infection is already established.

SUMMARY AND CONCLUSIONS

The effects of penetrating wounds of the brain by protruding foreign bodies and by deeply embedded foreign bodies have been studied in 54 dogs. The foreign bodies were unsterile and were inserted to varying depths and left in place for varying periods of time.

Foreign bodies penetrating the ventricle and allowed to remain protruding through the skin invariably produced a fulminating, fatal infection of meninges, brain and ependyma.

Removal of the protruding foreign body within 12 hours after its insertion greatly reduced the incidence of fatal infection.

Failure to penetrate the ventricle by a protruding foreign body considerably reduced the incidence of fatal infection.

Closure of the scalp over the inserted foreign body reduced the incidence of fatal infection and prolonged the survival time if infection developed.

Deeply embedded foreign bodies which did not communicate with the skin or subarachnoid space did not cause fatal infection unless the ventricle had been traversed.

In the presence of established superficial cerebral infection about protruding foreign bodies, early adequate drainage greatly reduced the mortality rate.

The following therapeutic suggestions may be made:

(1) Foreign bodies in the brain which are in communication with the skin, the subarachnoid space or the ventricular system should be removed at the earliest possible moment.

(2) Deeply embedded foreign bodies not falling in the category just mentioned should be removed only if focal irritation or destructive symptoms are present.

(3) If infection already exists about a superficially placed or protruding foreign body, its removal should be accompanied by the establishment of adequate open drainage.

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PULSATING TUMORS OF THE STERNUM

REPORT OF FOUR CASES

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INTEREST in the subject of pulsating tumors of the sternum was stimulated by the difficulties experienced in making the correct diagnosis in a case of hypernephroma of the right kidney which was recently seen at the Cleveland Clinic. In this case the only significant clinical or laboratory finding was a pulsating tumor of the sternum.

CASE I.—A man, 53 years of age, entered the clinic July 31, 1934, complaining of a pulsating lump over the sternum. This lump had been noticed ten months before admittance. The patient remembered that he had occasionally felt a "stinging sensation" over the sternum for nearly a year before the swelling appeared. During the ten months previous to this examination the tumor had slowly and painlessly enlarged and a distinct pulsation had developed.

The family history and the patient's past history had no bearing on the case. There was no history of lues. The patient had had no cardiac or pulmonary symptoms and there was no history of hematuria, frequency, nocturia or dysuria.

Physical examination showed a well developed and well nourished man, apparently in perfect health. The only positive finding was a round, firm, expansibly pulsating tumor about 4 cm. in diameter which apparently arose from the sternum at the level of its junction with the third costal cartilage. The overlying skin was freely movable but the tumor was fixed to the sternum. There was no discoloration of the skin and no dilatation of the superficial veins. A faint systolic bruit was audible over the tumor but no thrill could be felt. The kidneys were not palpable and there was no costovertebral tenderness.

A roentgenogram of the chest revealed no evidence of metastatic malignancy. Repeated urinalyses were negative with the exception of an occasional faint trace of albumen. No red cells were found in the examination of numerous specimens. Examination of the blood revealed 5,300,000 red cells; hemoglobin, 91 per cent; leukocytes, 8,600 with 80 per cent polymorphonuclear neutrophils, 18 per cent lymphocytes, and 2 per cent monocytes. The blood urea was 33 mg. per 100 cc., the blood sugar 109 mg. per 100 cc. and both the Wassermann and Kahn tests were negative.

A roentgenogram of the kidney, ureter and bladder region was made in an attempt to demonstrate a kidney tumor, but no pathologic enlargement of either kidney was noted.

The tentative diagnoses were (1) angiosarcoma; (2) metastasis from an hypernephroma; and (3) aneurysm of the internal mammary artery.

Since the tumor was apparently primary in the sternum, its operative removal was attempted and an encapsulated tumor 4½ cm. in diameter was identified as arising from the sternum. The surgeon was able to enucleate the tumor but this procedure was followed by a profuse hemorrhage which was controlled only by means of packs. Actual cautery with hot irons was then applied in order to obtain hemostasis and to eradicate any remaining traces of the tumor.

Pathologic Examination.—Metastatic hypernephroma.

The patient's postoperative course was uneventful except that the wound was slow in healing and a small area of granulation stubbornly persisted. A search for a primary tumor was instituted but an intravenous urogram on the sixteenth day after operation showed no evidence of a renal tumor and both kidneys were functioning normally. Shortly

before the patient's discharge from the hospital a slight pulsation was visible around the edges of the operative wound.

Since there was no obvious indication as to which kidney was involved, and in view of the pulsation of the thoracic wound, it was somewhat questionable as to whether or not tumor tissue was still present in the sternum, and no further operative procedures were recommended. Several weeks after his discharge from the hospital, the patient died as the result of an accident.

At necropsy an erosion of the sternum was found which was 2 cm. in diameter and was filled with soft yellow tumor tissue. There was a round yellow tumor 5 to 7 cm. in diameter which surrounded the right kidney pelvis. Sections of both these tumors showed a typical picture of hypernephroma. There was no evidence of any metastasis other than that to the sternum.

This interesting case raises a question as to the differential diagnosis of pulsating tumors of the sternum. The importance of the problem is quite obvious since the treatment of the various lesions which can give rise to pulsating tumors of the sternum differs so markedly. For example, confusion as to the diagnosis of an aneurysm, of a primary malignant tumor and of a secondary malignant tumor, could result in a needless fatality or in a useless operation. With this in mind, and with the hope of finding some diagnostic criterion which would be of value in separating the primary from the secondary pulsating neoplasms of the sternum, a review of the literature on pulsating tumors of the sternum has been made.

Sir James Paget,[1] in 1896, stated that "malignant disease of the chest wall receiving pulsation from the heart has been mistaken for aneurysm. The converse error has also been committed." Paget reported six cases of sarcoma of the sternum in none of which was pulsation present. Hedblom,[2] in an article on tumors of the bony chest wall, speaks of the difficulties in the differential diagnosis of sternal tumors and makes the following statement: "Pulsating sarcoma may simulate aneurysm." No mention of a pulsating sarcoma of the sternum was included in the description of Hedblom's cases. Heuer[3] collected 38 cases of tumor of the sternum from the literature; 61 per cent of these tumors he classified as primary sarcomata. Twelve per cent were primary cartilaginous tumors, one was a gumma and one a chronic inflammatory lesion. No tumor in this entire group showed pulsation. The seven remaining cases, in which a diagnosis was stated, were all cases of metastatic hypernephromata, and three of these seven showed pulsation. Heuer and Andrus[4] also collected a large number of cases of sternal tumor but they do not mention pulsation in any case. The most common origin of the metastatic tumors of the sternum in this series was carcinoma of the breast.

The remainder of the literature on sarcoma of the sternum and on pulsating tumors of the sternum was searched without finding any report of a case of verified pulsating sarcoma of the sternum. Hodgkin's disease, myeloma, tuberculous caries, as well as the above mentioned tumors are reported, but there is no reference to their pulsation or to any likelihood of their confusion with an aortic aneurysm.

In 1882, Rich[5] reported a case in which a pulsating tumor of the sternum was associated with a pyopericardium together with pyemia and multiple abscesses, but in this case the diagnosis was quite obvious. Cramer,[6] of von Langenbeck's clinic, reported a case of transmitted pulsation from a retrosternal tumor of thyroid origin. There was no suggestion in this case but that the origin of the tumor was mediastinal and not sternal. Rankin[7] reported one case and collected three others of true aneurysm of the internal mammary artery and Weiting[8] reported two cases of false (post-traumatic) aneurysm of this artery, but in all these cases the tumor appeared lateral to the sternum and the possibility that it arose from the sternum was never considered.

From this brief review of the literature, it is apparent that there is no reported case of a verified pulsating primary neoplasm of the sternum and that with the aid of modern facilities, there should be no difficulty in the diagnosis of pulsating sternal tumors which are due to aneurysms or other non-neoplastic disease.

PULSATING NEOPLASMS OF THE STERNUM

Collected from the Literature

Ten verified and three probable cases of pulsating neoplasm of the sternum have been reported. In five cases in the group in which the diagnosis was verified histologically, the tumors were metastases from hypernephromata of the kidney and in five the tumors were metastases from malignant adenomata of the thyroid. In the other three cases in which histologic studies of the tumor were not made, the description and the clinical course make it highly probable that the tumor was metastatic from a hypernephroma or from a very small and undetected malignant adenoma of the thyroid. No case of verified pulsating sarcoma of the sternum has been reported.

The verified cases of hypernephroma with pulsating metastases which have been reported in the literature are shown in Table I, the cases of probable hypernephroma with pulsating metastases to the sternum are shown in Table II and the verified cases of malignant tumors of the thyroid with pulsating metastases to the sternum are seen in Table III.

The designation of the sternal tumors described in Table III as metastases of malignant adenomata of the thyroid rather than as "benign metastasizing goiters" may at first glance seem somewhat arbitrary. Simpson,[21] who reviewed these cases carefully, came to the following conclusions: "Metastases of thyroid carcinomata are subject to great variability in microscopic appearance and may assume the structure of normal thyroid tissues, benign thyroid adenoma or simple colloid goiter." There is an abundance of evidence to indicate that there is no such entity as "benign metastasizing goiter" and that the use of this confusing term should be abandoned. Simpson is supported in this conclusion by the more recent studies of Dinsmore and Hicken[22] and others who have investigated this problem. The above designation of the

TABLE I

HYPERNEPHROMATA WITH PULSATING METASTASES IN STERNUM
VERIFIED BY NECROPSY OR BIOPSY

Author	Reference Number	Date	Age	Sex	First Symptom or Sign	Sternal Pain	Examination	Roentgen Diagnosis	Evidence of Primary Tumor	Clinical Diagnosis	Treatment	Confirmation of Diagnosis
McLeod and Jacobs	9	1921	54	M.	Tumor, upper sternum	None	Pulsating tumor 6 by 6 cm. in upper sternum	Sarcoma of sternum	None; urine negative	Sarcoma	None	Biopsy; hypernephroma
McLeod and Jacobs	9	1921	69	M.	Pain and tumor, sternum	Yes	Pulsating tumor 3½ by 6 cm. involving entire sternum	Erosion of sternum	None	Aortic aneurysm	None	Autopsy; tumor size chestnut in left kidney with metastases to sternum
Eshner	10	1908	60	F.	Tumor, upper sternum	None	Pulsating tumor 5 by 5 cm. upper sternum. Bruit audible	Aneurysm ruled out	None; urine showed only albumin and casts	Aortic aneyursm	None	Autopsy; tumor 4 by 5 cm. right kidney metastases to lungs, uterus and sternum
Dresser and Cabot Case	11 12	1923 1939	59	M.	Pain followed by tumor, upper sternum	Yes	Pulsating tumor 10 by 12 cm. upper sternum. Bruit heard	Malignant tumor of sternum	None; urine negative	(1) Aortic aneurysm. (2) Later primary neoplasm	Attempt at excision. X-ray	Autopsy; hypernephroma of kidney with metastases to ilium, sternum
Roth, Louis	13	1934	55	F.	Tumor, upper sternum	None	5 pulsating nodules 1½ by 1½ cm. upper sternum	Renal tumor	Hematuria occurred 1 year after appearance of tumor. Red blood cells in urine	Tumor of left kidney, vascular tumors of chest wall	X-ray	Autopsy; hypernephroma left kidney. Metastases to sternum and lungs. Cause of death, primary brain tumor

TABLE II

PROBABLE BUT UNPROVED CASES OF HYPERNEPHROMATA WITH PULSATING STERNAL METASTASES*

Author	Reference Number	Date	Age	Sex	First Symptom or Sign	Sternal Pain	Examination	Roentgen Diagnosis	Evidence of Primary Tumor	Clinical Diagnosis	Treatment	Confirmation of Diagnosis
Damourette	14	1890	56	F.	Tumor of upper sternum	None	Pulsating tumor 5 by 4 cm. upper sternum	None	None	(1) Aortic aneurysm. (2) Later sarcoma	None	Autopsy; metastases in ribs, ilium, femur, vertebrae, and sternum. No histologic examination. No mention made of thyroid or kidneys
Orsi	15	1893	62	M.	Pain and tumor in axilla followed by tumor upper sternum	None	Pulsating tumor of rib and upper sternum	None	None	Osteosarcoma	None	None
Ollivier	16	1868	53	F.	Tumor in upper sternum	None	Pulsating tumor 7 by 12 cm. upper sternum. Bruit heard. Pulsating tumor of skull. Paraplegia	None	None; unless primary tumor was in thyroid, a slight enlargement of which had been present for many years with recent growth	Aortic aneurysm, aneurysm of vessels of skull	None	Autopsy; limited. No mention of kidneys or thyroid. No histologic studies. Tumor had gray color and resembled "sarcoma or cancer of bone."

* In spite of the fact that in these cases no mention is made of a primary tumor in the kidney, it is quite possible, especially in view of very incomplete necropsy data presented, that a small hypernephroma could have been overlooked. In view of the rarity with which bone sarcoma metastasizes to bone, and taking into consideration the normal appearance of the thyroid, the long course of the disease, the absence of the localizing symptoms and signs which are usually associated with advanced carcinoma of organs other than the kidney and the absence of descriptions of the kidneys in the postmortem reports, it is quite likely that the primary tumors in these cases were hypernephromata.

TABLE III

MALIGNANT ADENOMATA OF THYROID WITH PULSATING METASTASES IN STERNUM VERIFIED BY NECROPSY OR BIOPSY

Author	Reference Number	Date	Age	Sex	First Symptom or Sign	Sternal Pain	Examination	Roentgen Diagnosis	Evidence of Primary Tumor	Clinical Diagnosis	Treatment	Confirmation of Diagnosis
Maingot	17	1926	73	F.	Tumor upper sternum	None	Pulsating tumor 7½ by 7½ cm. upper sternum. Dilated overlying veins	Destruction of manubrium	Two enlarged cervical nodes. Thyroid normal to palpation	None stated	None	Biopsy showed "malignant thyroid tumor"
Cramer	6	1887	49	M.	Pain in chest and left arm. Enlargement of neck	Yes	While in hospital following removal of goiter, pulsating tumor of upper sternum developed	—	Goiter	Aortic aneurysm	None	Autopsy; "Multiple metastases of malignant thyroid tumor"
Helbing	18	1901	51	F.	Tumor, upper sternum	None	Pulsating tumor 6 by 6 cm. upper sternum	—	Enlargement of right lobe of thyroid not noticed until years after development of sternal metastasis	Aortic aneurysm	None	Autopsy; malignant thyroid tumor with multiple metastases
Halbron	19	1904	68	F.	Goiter	—	Pulsating tumor 8 by 10 cm. upper sternum	—	Soft goiter; 4 years	Aortic aneurysm	None	Autopsy; apparent benign goiter; malignant thyroid tumor involving right sternoclavicular articulation
Carle	20	1897	50	F.	Tumor in sternum	None	Pulsating tumor in sternum	—	None	None stated	Excision	Autopsy; alveolar cancer of sternum. Many small metastatic nodules of typical thyroid structure in lungs*

* There is a possibility that this may have been a parathyroid tumor as postoperative tetany is mentioned. In view of the date (1897) this statement is probably inaccurate. No calcium studies were made and the pulmonary metastases showed definite thyroid structure.

sternal tumors reported in the older literature as metastases of benign goiters therefore seems justifiable.

PULSATING NEOPLASMS OF THE STERNUM

Cleveland Clinic Series

Four cases of malignant adenoma of the thyroid with pulsating sternal metastases have been seen at the Cleveland Clinic. These, with the case of hypernephroma reported above, make a total of five pulsating neoplasms of the sternum which have been observed in a series of 16 tumors of the sternum. These include five primary sarcomata, four metastases from carcinomata of the breast, four metastases from malignant adenomata of the thyroid, one cavernous hemangioma, one chondroma and one hypernephroma. The only tumors that showed pulsation were those which had metastasized from malignant adenomata of the thyroid and from the hypernephroma. This again emphasizes the rarity with which pulsation is observed not only in primary tumors of the sternum, but also in metastatic carcinomata other than hypernephromata or malignant adenomata of the thyroid.

The following cases are those in which metastasis occurred from a malignant tumor of the thyroid gland.

CASE REPORTS

CASE II.—The patient was a woman, aged 63 years. A tumor of the thyroid presenting the typical gross and histologic features of a malignant adenoma had been removed in 1924. Four years later a firm, fixed, painless mass developed over the manubrium. Roentgen examination revealed destruction of bone. A diagnosis of sternal metastasis from a malignant adenoma of the thyroid was made and after the administration of roentgen therapy the patient improved. She was not seen again for two years at which time the mass over the sternum exhibited pulsation, and it was found that metastasis to the ilium had occurred. In 1932, eight years after the thyroidectomy, the patient died with generalized metastases. (Classified as malignant adenoma of the thyroid with multiple metastases including the sternum, verified.)

CASE III.—The patient was a man, 57 years of age, in whom one year before admission a goiter had been partially removed, the surgeon having been forced to stop because of hemorrhage. A biopsy was taken February 14, 1919, which the pathologist reported as presenting the typical histologic features of a malignant adenoma. Three months after the biopsy the patient returned and examination revealed a pulsating tumor of the sternum, a biopsy of which showed the presence of malignant adenoma. (Classified as malignant adenoma of the thyroid with metastasis to the sternum, verified.)

CASE IV.—The patient was a man, 63 years of age, who was first seen at the clinic in October, 1930. He complained of recent enlargement of a goiter which had been present for 35 years. October 3, 1930, a papillary adenoma of the thyroid of questionable malignancy was removed. Four years later it was found that he had a recurrent tumor of the thyroid, a large pulsating tumor of the ilium, destructive lesions involving the cervical vertebrae, and a pulsating tumor involving the upper part of the sternum. A puncture biopsy was reported by the pathologist as showing colloid-containing follicles and a structure suggestive of a malignant adenoma of the thyroid. A diagnosis of recurrent and metastatic malignancy of the thyroid was made. (Classified as malignant adenoma of the thyroid with multiple metastases including the sternum, verified.)

CASE V.—The patient was a man, 68 years of age, who entered the clinic July 2, 1930, complaining of pain in the right hip and the left shoulder. Examination revealed

pulsating tumors of the sternum and of the ilium. A systolic bruit was audible over these tumors. There was a hard adenoma of the thyroid isthmus, 2 cm. in diameter, and a second nodule was present at the lower pole of the right lobe of the thyroid. A roentgenogram showed metastatic carcinoma in the mediastinum, ilium and lungs. Dr. Allen Graham, who saw the patient in consultation, made a diagnosis of malignant adenoma of the thyroid with multiple metastases. (Classified as probable malignant adenoma of the thyroid with multiple metastases including the sternum, unverified.)

In addition to the above cases of pulsating sternal tumors, two pulsating tumors of the inner end of the clavicle have been seen at the clinic. One of these was metastatic from a malignant adenoma of the thyroid while the other was secondary to a hypernephroma.

COMMENT

In this total series (collected and reported) of 18 pulsating neoplasms of the sternum, nine were probably metastatic from hypernephromata of the kidney and nine were probably metastatic from malignant adenomata of the thyroid.

The average age of the patients in whom hypernephroma was present was 58 years and of those with malignant adenoma 60.2 years. The ratio of men to women was 5 to 4 among those with hypernephroma and 4 to 5 among those with malignant adenomata of the thyroid.

In eight of the nine cases of hypernephroma, sternal pain or the appearance of a tumor over the sternum was the first evidence of the disease. In no case were any urinary symptoms or signs observed until after the appearance of the pulsating sternal tumor and in only one case did urinary symptoms appear before the patient's death.

In only four cases of malignant adenoma was the appearance of a tumor or of pain over the sternum the leading symptom. In the other cases, the first symptoms were caused by the primary tumor or by metastasis to regions other than the sternum. In six of the nine cases, there was clinical evidence of the primary thyroid tumor at the time of the first examination.

In every case of malignant adenoma and of hypernephroma, the tumor occupied the upper part of the sternum, with the exception of one case in which the entire sternum was involved. The tumor was usually described as of a firm consistency although the terms "elastic" and "semifluctuant" were occasionally applied to the description of its consistency.

In four of the nine cases of hypernephroma, the diagnosis before biopsy or postmortem was aortic aneurysm. A diagnosis of pulsating primary sarcoma was made in the other five cases.

A diagnosis of aneurysm was made in three of the nine cases of malignant adenoma. No diagnosis was mentioned in two cases. In the remaining four cases the correct preoperative diagnosis was made.

In all nine cases in which appropriate roentgenograms were made, aortic aneurysm was ruled out as the cause of the tumor.

In each group of cases the metastasis was single three times and multiple six times. The survival period after the appearance of the first symptom

averaged 35.9 months in the group in which hypernephromata were present and 32.4 months in the group with malignant adenoma. One patient in whom a malignant adenoma was present survived 17 years after the appearance of the metastatic sternal tumor, although no treatment was given. The average survival period of the three patients treated with roentgen ray was 61 months as compared with 41 months for the patients in whom the sternal tumor was incompletely excised or who had no treatment at all (Table IV).

TABLE IV

PULSATING TUMORS OF THE STERNUM

	Hypernephroma 9 cases	Malignant Adenoma 9 cases
Average age	58 years	60.2 years
Males	55.5 per cent	45.5 per cent
Evidence at time of appearance of sternal tumor indicating the presence of the primary tumor.	0 per cent	77.8 per cent
Final clinical diagnosis		
(*a*) Correct	0 per cent	44.4 per cent
(*b*) Aortic aneurysm	44.4 per cent	33.3 per cent
(*c*) Primary sarcoma	55.6 per cent	0 per cent
(*d*) No diagnosis stated	0 per cent	22.2 per cent
Single metastasis	33.3 per cent	33.3 per cent
Survival after first symptom	35.9 months	32.4 months

A review of the literature on the subject of the skeletal metastases of hypernephromata and malignant adenomata of the thyroid indicates that these two types of tumor are almost unique in their tendency to produce slow growing, pulsating and often single skeletal metastases. Hence it is not surprising that these two tumors are the only neoplasms which have been reported as producing pulsating tumors of the sternum.

Pulsation is rarely present in sarcomata, and the sternum is an unusual site for the development of a sarcoma. It is therefore not remarkable that no report of a verified case of pulsating sarcoma of the sternum is to be found in the literature.

The metastases from carcinomas of the breast (the only other metastatic tumor which frequently involves the sternum), have not been reported as showing pulsation. Aneurysms of the internal mammary artery and other rare conditions giving rise to pulsations in this vicinity have not been confused with neoplasms of the sternum. Therefore, the only difficulty in the clinical differentiation of pulsating tumors of the sternum is in distinguishing between aortic aneurysms and metastases from hypernephromata or from malignant adenomata of the thyroid. If aortic aneurysm is ruled out by roentgenologic studies of the mediastinum and if examination of the thyroid does not suggest a malignant change, the sternal tumor is in all probability a metastasis from a hypernephroma and pyelograms should be made. Finally it should be remembered that even if these are negative, the tumor may still be a hypernephroma, for the primary tumor is often very small.

TREATMENT.—No report could be found in the literature of a case of hypernephroma with a single metastasis or of a malignant adenoma of the

thyroid with a single metastasis in which both the primary and the secondary tumor had been removed and the patient cured. Numerous cases have been reported in which the metastatic tumor has been mistaken for a primary sarcoma and a radical resection has been performed. The patient reported by Albrecht[23] lived for ten years after the resection of a metastatic hypernephroma of the scapula. Geschickter and Copeland's[24] patient received radium treatment for a metastatic hypernephroma and was alive more than 12 years after the onset of symptoms. Roentgen therapy apparently held the growth in check in the case reported by Dresser[11] and the patient lived seven years and four months after the onset of symptoms and five years after partial extirpation of the sternal tumor, followed by roentgen therapy.

Despite these apparently favorable reports of the results obtained in the treatment of these metastatic tumors, it must be remembered that adequate surgical treatment of a sternal metastasis should involve resection of the sternum, an operation which is not without hazard. Hypernephromata, moreover, are usually considered to be quite resistant to irradiation. Roentgen or radium treatment can doubtless temporarily benefit the majority of patients with metastases from thyroid tumors, but it is not certain that either can effect a permanent cure. The apparently favorable results of such treatment must be viewed critically when one remembers that in the case reported by Helbing,[18] the patient lived 17 years after the sternal metastasis was first noticed and that this patient received no treatment of any kind.

In view of the naturally slow development of the disease, it is therefore probable that the more conservative treatment (irradiation) is preferable. If, however, the primary tumor is operable and if the patient is willing to accept the hazard of an attempt at complete extirpation both of the primary tumor and of the single metastatic lesion, there is apparently a possibility that this hitherto unreported procedure might result in a permanent cure. The only difficulty is in ruling out the presence of undetectable generalized metastases which, if present, would make the operation a futile procedure.

SUMMARY

(1) The importance of the correct preoperative diagnosis of pulsating tumors of the sternum is emphasized.

(2) Aortic aneurysm can be differentiated from a neoplasm of the sternum by roentgenographic examination of the mediastinum.

(3) No verified case of pulsating primary neoplasm (sarcoma, *etc.*) of the sternum could be found in the literature.

(4) Thirteen cases of pulsating neoplasm of the sternum have been reported in the literature and five more are added from the records of the Cleveland Clinic. Nine of these were probably cases of metastatic hypernephromata and nine were probably metastases from malignant adenomata of the thyroid.

(5) If a pulsating tumor of the sternum is not an aortic aneurysm, it is probably a metastasis from a malignant adenoma of the thyroid or from a hypernephroma.

(6) Resection of a pulsating neoplasm of the sternum should never be performed until every effort has been made to demonstrate the presence of a primary hypernephroma or of a malignant adenoma of the thyroid.

(7) The possibility of successful resection both of the primary tumor and of its single metastasis is discussed.

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CHOLANGIOGRAPHY

Visualization of the Gallbladder and Bile Ducts During and After Operation

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The uncertainty of diagnosis in diseases of the gallbladder and bile ducts contributes to the difficulties encountered in their surgical management. Our present diagnostic methods usually detect a diseased gallbladder but unfortunately they give but meager information concerning the provocative factors. For example, a cholecystogram merely evidences a normal or nonfunctioning gallbladder without revealing whether the disease is within the liver, the bile ducts or the gallbladder itself. The common bile duct may be occluded by a stricture, neoplasm, calculus, by extrinsic pressure, or from a spasm of its sphincter, and there is no way, other than exploration, of determining the causative agent. Even the direct operative examination is not infallible, for occasionally the surgeon fails to detect an elusive common duct stone; he may not recognize the physiologic blockade caused by a spasm or dyssynergia of the choledochal sphincter; he may be unable to demonstrate a pathologic obstruction to account for the dilated but otherwise normal appearing gallbladder; and he may even overlook small strictures or neoplasms. Hence, studies which facilitate the recognition of abnormalities that may interfere with the function of the liver and its ductal system are worthy of investigation.

Direct roentgenographic visualization of the biliary tree is a valuable diagnostic and therapeutic procedure. The injection of such radiopaque substances as lipoiodine directly into the gallbladder and bile ducts gives an accurate graphic picture of conditions as they really are. Diagnostically, it demonstrates whether the ductal system is patent or occluded; it notes the position and number of calculi; it defines the extent and location of strictures; it denotes the functional status of the sphincter of Oddi; it outlines fistulous communications; and it demonstrates dilatation and sacculation of the bile ducts. It also has definite therapeutic value for it enables one to determine how long the dilated biliary tract should be drained; it confirms the patency of the common duct before the drainage tubes are removed; it demonstrates whether a cholecystostomy will decompress the entire biliary

system; it helps to ascertain the cause of persistent external biliary fistulae; and occasionally the lipoiodine lavage will flush out small stones, sand, or mucous plugs that are causing the patient postoperative pain and discomfort. Any test, therefore, which graphically outlines the gallbladder and the minute ramifications of the bile passages is certain to reduce diagnostic and therapeutic errors.

The urologists were quick to develop the visualization studies of the genitourinary tract, thereby converting a haphazard problematical subject into one of the most exact specialties of medicine. We can profit by their example, for much information can be obtained by making graphic studies of the gallbladder and bile ducts. The technic and value of such cholangiographic observations are presented.

TECHNIC

There are two methods by which cholangiography, or visualization of the biliary system, can be accomplished. One plan, to be known as immediate cholangiography, consists of injecting some radiopaque substance such as lipoiodine into the gallbladder and bile ducts during the operation and making roentgenographic observations while the patient is still on the operating table. The other procedure, designated as delayed cholangiography, utilizes the drainage tubes, which were inserted into the gallbladder and biliary radicals at the time of operation, or the external biliary fistulae, for the introduction of the contrast solution. These visualization studies of the hepatic system may be made at any desired time. The indications for and value of each method will be emphasized by case presentation.

Lipoiodine is an efficient, nontoxic, nonirritating substance which gives an excellent graphic reproduction of the biliary system. No unfavorable systemic reactions were observed in spite of the fact that this contrast medium was injected into inflamed gallbladders, into infected bile ducts, and was used in the presence of stones, strictures, and even pancreatitis. The stock lipoiodine is a rather viscid substance; therefore, we have been diluting it to 33 1/3 and 50 per cent of its original concentration by adding sterile olive oil. The resultant mixture, when heated to body temperature, forms a labile solution, which readily flows into the smaller biliary radicals. Further dilutions are not advisable as the solutions then lose their contrast values. Stereoscopic roentgenograms combined with fluoroscopic studies yield much more detailed information than does a single flat film of the liver area.

Immediate Cholangiography.—Immediate cholangiography is particularly indicated in those problem cases in which the diagnosis is questionable or in which the selection of the proper surgical procedure is difficult. Although our experience has been limited to a comparatively few cases, we feel that it has definite merit. The technic is rather simple. The gallbladder is first exposed and the entire ductal system is carefully examined for abnormalities. The gallbladder is then collapsed by aspirating its bile contents, following which 40 to 60 cc. of warm lipoiodine are slowly injected into the

gallbladder lumen through the aspirating needle. If the cystic duct is patent, the contrast solution immediately enters the choledochus and outlines the entire ductal system. By means of a portable roentgen ray unit, roentgenograms are taken immediately, and if anatomic abnormalities are noted, the surgeon can proceed with the proper corrective measures.

Whitaker[15] demonstrated that the gallbladder and bile ducts fill only when the choledochal sphincter is closed. The anesthetic, particularly spinal anesthesia, may cause the sphincter of Oddi to relax and permit the lipoiodine to flow unhindered into the duodenum, thus failing to outline the small biliary

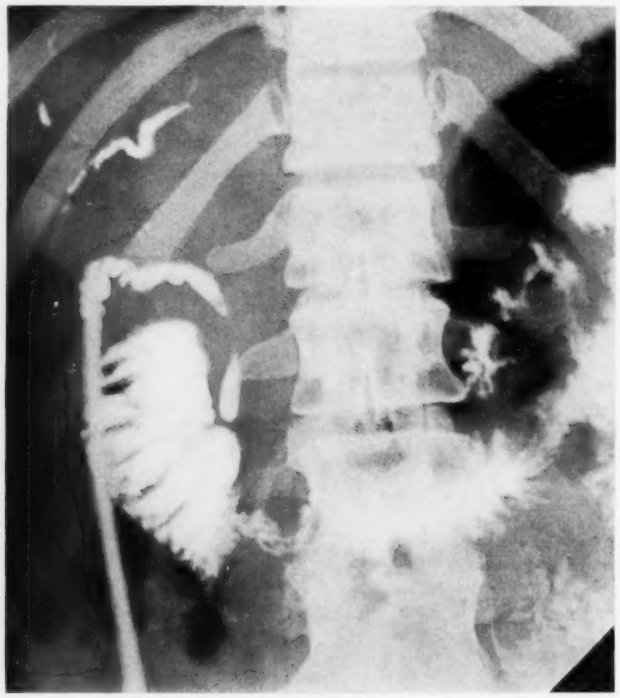

FIG. 1.—(Case I.) Immediate cholangiography. Forty cc. of lipoiodine were introduced into the common duct via the stump of the cystic duct. The oil immediately escaped into the intestinal tract, proving the choledochus to be patent. As there were no abnormalities at the ampulla, there was no need to explore the common bile duct. The tube was removed, the cystic duct was ligated and the abdomen closed.

radicals. In such an event, if further studies are necessary the intestinal orifice of the common bile duct can be occluded by placing a small gauze pack against the second portion of the duodenum and the bile ducts again injected with the radiopaque material. We are now investigating the influence of anesthetics on the choledochal sphincter.

After removing the gallbladder, the surgeon may desire to test the patency of the common duct before closing the abdomen. This can easily be accomplished by injecting the lipoiodine directly into the choledochus, or through the stump of the cystic duct. If the visualization studies fail to show the pres-

ence of strictures, neoplasms, stones or dilatation of the bile ducts, and if the contrast solution enters the intestinal canal, then the common duct need not be explored or drained. Mirizzi[9] was the first to make a practical application of immediate cholangiography.

Case I.—Mrs. A. A., housewife, aged 29, entered the hospital with the chief complaint of "gallbladder pains." Following her first pregnancy two and one-half years ago, she had had recurrent attacks of sharp colicky pains in the right hypochondrium associated with nausea, vomiting, jaundice and acholic stools. Physical examination and laboratory studies were normal with the exception of two cholecystograms which demonstrated a poorly functioning, dilated gallbladder.

At operation the gallbladder was found to contain three small stones and was distended with a golden brown bile. The walls of the cystic duct were indurated, edematous and fibrosed. The common and hepatic bile ducts were slightly dilated but otherwise normal. The head of the pancreas was firm, indurated and slightly enlarged.

After performing a cholecystectomy, a small rubber catheter was inserted into the stump of the cystic duct and 40 cc. of lipoiodine were injected into the choledochus. The bile ducts were not visualized because the oil immediately entered the duodenum (Fig. 1). The indurated swollen pancreas was not acting as a mechanical obstruction to the flow of bile; therefore there was no indication for common bile duct drainage. The catheter was removed, the cystic duct was ligated and the patient made a very rapid convalescence.

Cholangiography is practical because of the ease with which such studies are performed. If one does not desire or is unable to insert a catheter into the stump of the cystic duct, the contrast media can be injected directly into the common duct by means of a needle, and then the necessary radiographs made. Had we been interested in visualizing the entire ductal system in this patient, it would have been necessary to occlude the duodenal end of the common duct by pressure on the second portion of the duodenum and then to reinject the lipoiodine. In this particular instance, however, we were merely interested in the patency of the duct at the ampulla because of the associated pancreatitis. These visualization studies do not increase the time required for operation more than ten minutes.

Delayed Cholangiography.—In delayed cholangiography, or the postoperative visualization study of the extrahepatic bile ducts, the lipoiodine is injected into the drainage tubes which were sutured into the gallbladder or biliary ducts at the time of operation, or into the external biliary fistulae. Roentgenograms are taken immediately after the oil has been introduced, thus obtaining an exact outline of the biliary system. If any abnormalities are noted, then serial roentgenograms are taken at 15 minute intervals until the diagnosis is established. The value of these studies can best be demonstrated by presenting actual cases.

(*a*) *Determining the patency of the cystic duct.*—The therapeutic value of a cholecystostomy does not entirely depend on the simple drainage of the diseased gallbladder but is also dependent upon the decompression and drainage of the entire hepatic system. If the cystic duct is occluded prior to or subsequent to the operation, then the value of a cholecystostomy is definitely minimized, for the bile ducts and hepatic radicals are not drained. It is true that the inflammatory occlusion of the cystic duct usually disappears after draining the infected gallbladder for a week or ten days. It is, how-

ever, during this crucial period that the entire ductal system should be drained so as to facilitate the reestablishment of hepatic functions and to reduce the high morbidity and mortality rates of acute cholecystitis and cholangitis. One cannot accurately determine the functional status of the cystic duct by direct observation and palpation. Immediate cholangiography, or injection of lipoiodine into the gallbladder, will readily test the patency of the cystic duct, but one hesitates to utilize the visualization studies in the presence of acute infections for fear of disseminating the inflammatory process. Thus far, however, we have had no unfavorable reactions following lipoiodine injections in seven cases of cholangitis. The value of cholecystostomy and choledochostomy in cases of generalized suppurative process of the biliary tree is problematical and will require additional investigation. One cannot forget that the gallbladder is but a small part of the biliary system and that if the cystic duct is occluded a cholecystostomy merely drains a small segment of the infected area. Might not the high morbidity rate following the drainage of the gallbladder be due to a retention of infected bile in the large bile ducts and hepatic radicals? Investigations are now being carried out, hoping thereby to answer some of these questions.

CASE II.—J. H., farmer, 53 years of age, was admitted to the hospital because of "indigestion." During the past four years he had been annoyed by the belching of gas, the passage of flatus, and epigastric distress. Occasionally, the colicky pains became quite severe. He denied ever having been jaundiced.

On examination the skin was found to have a definite icteric tinge. In the right hypochondrium there was a firm, ovoid, fixed, tender mass. The liver could not be differentiated from the tumor and the gallbladder was nonfunctioning according to the cholecystogram.

At operation the gallbladder was found to be moderately distended. Its walls were thickened, indurated and scarred. The liver appeared normal except for the fibrosis of its capsule. The common duct was about twice its usual size, and in its retroduodenal portion there was a small lobulated mass which was thought to be either an enlarged lymph node or an incarcerated intramural calculus. Exploration of the choledochus disclosed a firm annular stricture just distal to the junction of the hepatic and cystic ducts. This cicatricial band had reduced the lumen of the common duct to one-fourth its original size. The location and extent of the stricture precluded the possibility of its excision for the continuity of the bile ducts could not be reestablished, nor could a hepatico-enterostomy be done. After demonstrating that the duodenal portion of the common duct was patent a cholecystostomy and choledochostomy was performed. This conservative procedure not only decompressed the biliary system but it left the gallbladder intact so that a cholecysto-enterostomy could be performed at a later date should the inflammatory stricture again occlude the common duct.

While scooping out the calculi from the gallbladder, about 20 cc. of brownish yellow bile escaped, indicating that the cystic duct must be patent. A large rubber tube was firmly anchored into the gallbladder for drainage purposes. The stricture in the common duct was divided longitudinally and a T-tube was inserted in such a manner that one limb of the T would dilate the constricted portion of the duct. Much to our surprise, for we had thought that the cystic duct was patent, no bile drained from the cholecystostomy tube. Apparently all the bile could find an easy exit by the T-tube or else the cystic duct had become occluded. In order to answer this question, the following studies were made.

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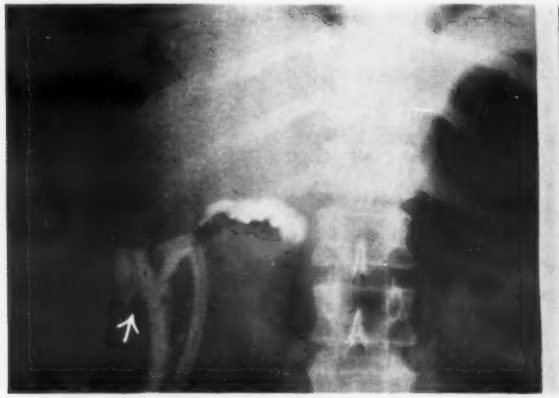

FIG. 2.—(Case II.) Forty cc. of lipoiodine were injected into the gallbladder through the cholecystostomy tube. The gallbladder was only partially visualized as the oil escaped around the drainage tube. Stereoscopic studies indicated that the occluded cystic duct had prevented the lipoiodine from entering the common duct. The arrow points to a collection of oil outside of the choledochus. During the operation it was thought that the cystic duct was patent, but cholangiographic studies demonstrated a postoperative occlusion of this duct.

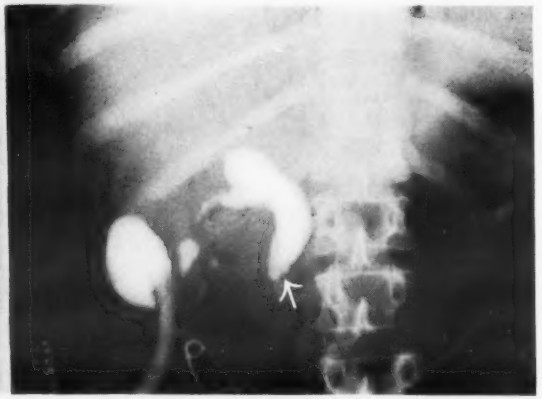

FIG. 3.—(Case II.) Sixteen days later the visualization studies were repeated. The gallbladder was filled with oil, the cystic duct was patent, and the lipoiodine had filled the dilated common duct. Note the obstruction at the terminal portion of the choledochus. Progressive roentgenographic observations demonstrated this to be a spasm of the choledochal sphincter.

Forty-eight hours after the operation, 40 cc. of lipoiodine were injected into the cholecystostomy tube. Roentgenograms were taken immediately and again in 20 minutes. In both instances, the lipoiodine was confined within the gallbladder for it had been unable to traverse the cystic duct (Fig. 2). This seemed to contradict the operative findings for it must be remembered that apparently normal bile was found in the gallbladder, indicating the cystic duct to be open. Evidently the operative trauma had initiated sufficient tissue reaction to produce an inflammatory obstruction of the cystic duct.

This observation teaches the object lesson that a cholecystostomy does not always decompress and drain the entire biliary system. If a patient should have an associated cholangitis and bile stasis due to choledochal obstruction, then the mere drainage of the gallbladder would not relieve the intraductal pressures or permit the infected bile to escape. In this particular instance, it was 16 days before the blockade was removed so that bile and lipoiodine could pass unhindered through the cystic duct (Fig. 3). Fortunately, the T-tube in the common duct had supplied adequate drainage of the hepatic radicals and facilitated the recovery of the patient. It is conceivable that the unfavorable response of some patients with acute empyema of the gallbladder to a simple cholecystostomy might be due to the inflammatory occlusion of the cystic duct. Visualization studies at the time of operation or during the convalescence of the patient will help determine the patency of these structures. In this small series of 11 cases, we encountered two instances in which the roentgenographic studies demonstrated a complete occlusion of the cystic duct. In both instances the common bile duct was drained, permitting the infected bile to escape, and thus facilitating the recovery of the patients.

(*b*) *Determining the cause of persistent external biliary fistulae.*—Since the work of Lanari and Squirri[6] in 1924, visualization of bile ducts by introducing lipoiodine into the external fistula has become an established diagnostic procedure. The contrast media not only locates the obstructive agent but it gives information which is invaluable in selecting the operation best suited to correct the existing abnormalities.

Case III.—L. B., farmer, 56 years of age, complained of "gall running from his side." Four months previously he had had a cholecystostomy performed elsewhere, at which time several small stones were removed from the gallbladder. He had a stormy convalescence associated with chills, fever, and a persistent drainage of bile from the operative area. During the past six weeks the flow of bile had become intermittent. During the period of free drainage he felt fine, but when the external sinus became occluded he had severe colic and epigastric pains. The major portion of bile was passing into the intestinal tract as his stools were of normal color and he had no evidence of jaundice. Apparently he had a partial obstruction in the cystic duct which permitted bile to flow into the gallbladder but prevented it from returning to the common duct.

In order to obtain a graphic outline of the biliary system, 60 cc. of lipoiodine were injected into the external biliary fistula. Roentgenograms were made immediately. The gallbladder was well visualized and no calculi were seen. The distal portion of the cystic duct was definitely outlined, showing an irregular spiral-shaped shadow. It was impossible to say whether this shadow represented hypertrophied spiral valves of

Heister, an inflammatory swelling of the cystic duct, or several small stones. The distal end of the cystic duct was apparently occluded for the warm lipoiodine did not flow from the gallbladder into the common bile duct in spite of being injected under positive pressure. The lipoiodine was then diluted with twice its volume of olive oil, the resulting mixture heated to body temperature and injected into the fistula, but even this more labile solution did not enter the common duct. It seemed highly improbable that the cystic duct was completely occluded, for bile had been draining from the fistula but 24 hours previously. A second roentgenogram made 30 minutes later showed that a very small amount of oil had slowly trickled into the duodenum (Fig. 4). From these visualization studies it was concluded that the external biliary fistula was caused by a partial occlusion of the cystic duct.

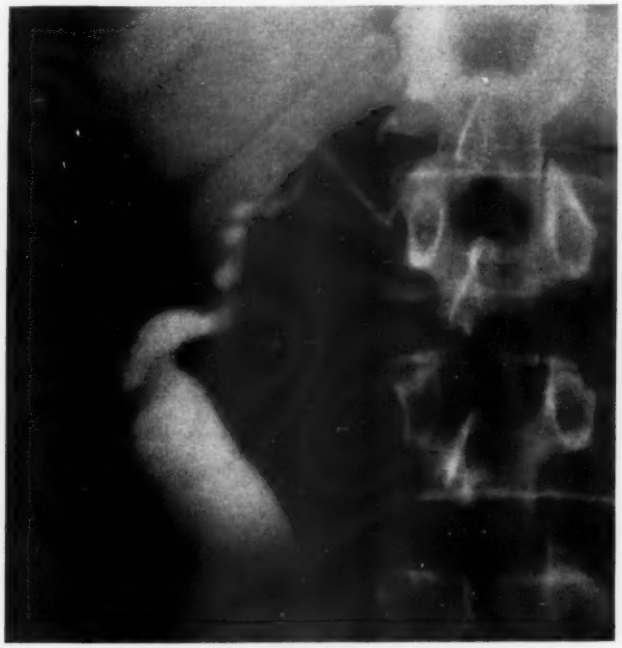

FIG. 4.—(Case III.) Sixty cc. of lipoiodine were injected into the external biliary fistula. The gallbladder appeared normal. The proximal three-fourths of the cystic duct was visualized but exhibited some peculiar configurations that were taken to be the spiral valves of Heister. The distal one-fourth of the duct was occluded. An organized bile stained mucous plug was found to obstruct the cystic duct and it protruded into the neck of the gallbladder. Bile could enter the gallbladder but the ball valve action of the clot prevented its return through the cystic duct, hence the external fistula.

Operative intervention confirmed this diagnosis, for a long, well organized, bile stained mucous plug filled the entire cystic duct and projected into the neck of the gallbladder. This plug was firmly anchored to the walls of the cystic duct and had a spiral configuration, thus accounting for the peculiar roentgenographic outline. Along the anterior surface of the plug was a small groove which permitted bile to enter the gallbladder, but the ball valve action of the intravesical portion precluded its escape. A cholecystectomy was performed, the wound healed by primary intention, and the patient made an excellent recovery.

CASE IV.—Mrs. A. M., housewife, 50 years of age, was admitted to Doctor Hunt's service because of a persistent external biliary fistula. Seven years ago a cholecystectomy had been performed. The wound refused to heal and there had been a continuous

drainage from the fistulous tract. At times her stools were acholic but as a rule they had a brownish color. Five months prior to admission, the fistula healed spontaneously. She then became jaundiced, her stools were acholic and she had considerable epigastric distress. Two months later the sinus tract opened and there was a copious discharge of bile. She immediately felt improved, but the jaundice did not disappear.

Examination revealed a poorly nourished, jaundiced, asthenic woman. There was a continuous discharge of mucopurulent bile from the fistulous tract. The liver was slightly enlarged. A radiograph of the abdomen revealed an indefinite opacity just to the right of the second lumbar vertebra. This was interpreted as being a stone in the common duct.

The instillation of 20 cc. of lipoiodine into the external fistula demonstrated a dilated

FIG. 5.—(Case IB.) Twenty cc. of lipoiodine were injected through a biliary fistula of seven years' duration. Multiple small calculi could be seen along the fistulous tract and a stone measuring 2 cm. in diameter practically occluded the ampulla. Note the enormous dilatation of the bile ducts. This roentgenogram gives definite evidence why the biliary fistula did not heal.

sinus tract containing several small stones. The hepatic ducts and choledochus were of enormous size, the latter measuring 20 Mm. in diameter. The terminal portion of the common bile duct was obstructed by a large stone measuring 10 by 15 Mm. The mechanical blockade was not complete as a small trickle of lipoiodine managed to seep around the calculus (Fig. 5).

At operation several small stones were removed from the fistulous tract and four calculi from the choledochus. After extracting the large stone from the ampulla of Vater, the common duct was found to be patent. A small catheter was then anchored into the cystic duct for drainage purposes. The patient developed a severe suppurative

cholangitis and died one week after operation. These studies merely emphasize the value of cholangiography in determining the etiology of persistent biliary fistulae.

(*c*) *How long shall we drain the common duct?*—Cicatricial and calculous obstructions of the common bile duct are not dangerous in and of themselves but they effect their damage by indirectly diminishing the functional capacity of the liver. These obstructive agents cause an accumulation of bile resulting in a cylindrical dilatation of the biliary radicals, pressure atrophy of the hepatic cells, interference with the portal circulation, and if there be a coexisting infection, then a cholangitis, hepatitis and liver insufficiency follow.

Restoration of liver function then largely depends on removing the obstruction and controlling the infection. Ravdin[12] maintains that choledochostomy tubes should not be removed until the bile becomes microscopically and chemically normal, otherwise there is a retardation of the reparative processes. Thorlakson and McMillan[13] demonstrated roentgenographically that prolonged drainage of infected bile ducts resulted in a disappearance of the cholangiectasia and a cessation of the infection. In order to determine the effects of prolonged drainage of the choledochus, progressive visualization studies were made over 33-day periods in several patients, a typical case of which is presented.

Case V.—C. M., farmer, aged 69, came to the hospital because of "stomach trouble." For the last 35 years he had had considerable epigastric distress, characterized by bloating, passage of flatus, and gaseous eructations. He had suffered from recurrent attacks of jaundice, at which times his stools were acholic and his urine contained bile. He had lost 55 pounds of weight during the past ten months.

On examination a large, firm, fixed tumor was found in the epigastric area. The liver, spleen, and kidneys were not palpable. His skin and sclera were definitely jaundiced. Roentgenograms demonstrated a nonfunctioning gallbladder and several small calculi which were thought to be in the common bile duct. During the preoperative period, he had several chills and a temperature of 106° F. which indicated a cholangitis secondary to an intermittent calculous obstruction. The large tumor mass, the rapid loss of weight, and the persistent jaundice made the diagnosis of pancreatic malignancy associated with cholelithiasis seem plausible.

At operation the liver was found to have a dark green nutmeg color. It was slightly enlarged and its capsule was tense and fibrotic. The gallbladder was about twice its usual size and its walls were indurated and edematous. The cystic duct appeared to be patent. The choledochus was dilated to about three times its regular diameter and after aspirating 60 cc. of black, viscid bile, several stones could be palpated in the hepatic and common ducts. The pancreas was enlarged, firm and swollen but it did not present the stony hardiness of a malignancy. The gallbladder was removed and several stones were extracted from the hepatic ducts. Painstaking examination failed to show any gross obstruction at or near the ampulla of Vater. A T-tube was anchored in the common duct.

Visualization studies were deferred until the twelfth postoperative day because of acute cholangitis. Then 40 cc. of lipoiodine were permitted to flow into the T-tube. Roentgenograms which were taken immediately showed a definite dilatation of the common duct. The hepatic ducts and small biliary radicals were not visualized because the coexisting cholangitis and relaxed sphincter of Oddi had permitted the oil to drain into the duodenum (Fig. 6). On the twenty-ninth (Fig. 7) and again on the thirty-third (Fig. 8) postoperative days, visualization studies were made. The bile ducts were no

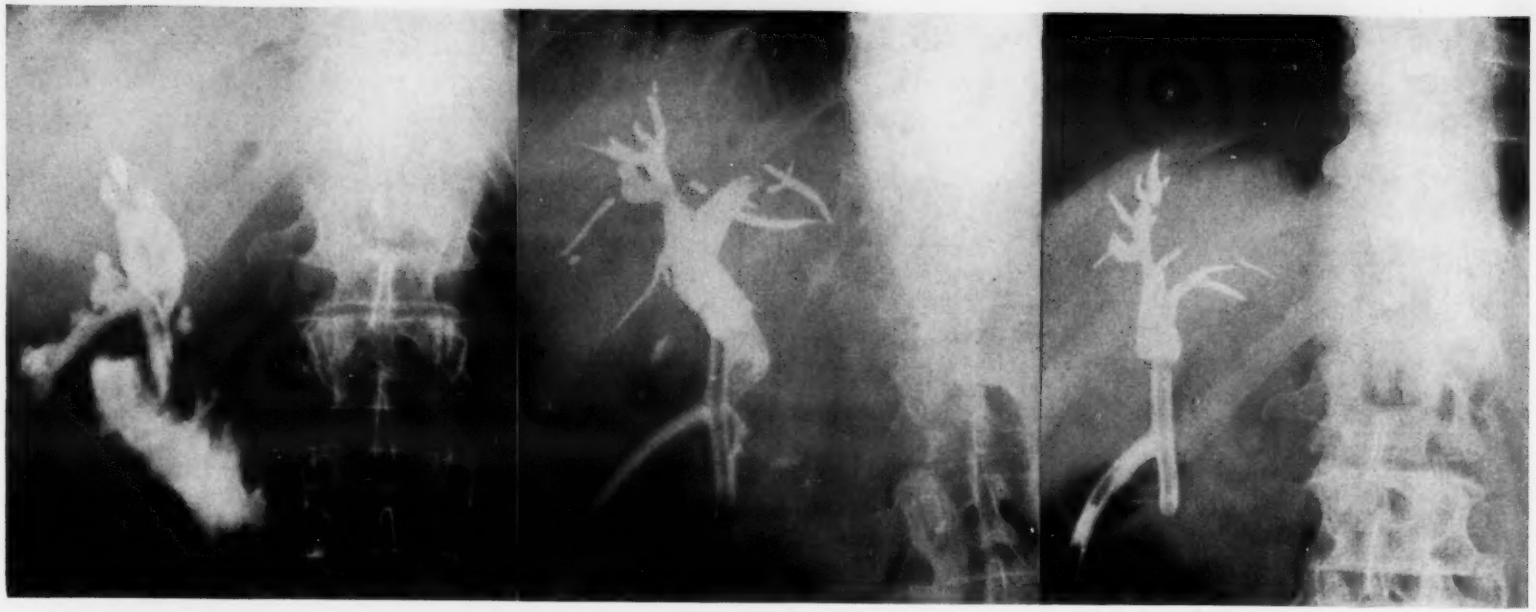

FIG. 6.—(Case V.) On the twelfth postoperative day, 40 cc. of lipoiodine were injected into the common duct. Note that the choledochus is dilated and that its relaxed sphincter permitted the oil to enter the duodenum in spite of a coexisting pancreatitis and cholangitis. Apparently inflammation of the common bile duct and adjacent structures does not necessarily produce a choledochal spasm.

FIG. 7.—(Case V.) Lipoiodine studies on the twenty-ninth postoperative day. The common bile duct is much smaller than before and the small biliary radicals are visualized, indicating a subsidence of the inflammatory process.

FIG. 8.—(Case V.) Cholangiogram made after 33 days of continuous drainage of the common bile duct. Note that the bile ducts are no longer dilated and that the inflammatory process has subsided, permitting the small hepatic radicals to be visualized. Comparison with Figs. 6 and 7 demonstrates the value of prolonged drainage of infected and dilated biliary trees.

longer dilated and the small radicals were patent, the graphic pattern of the entire biliary tree was practically normal. It was apparent that the prolonged drainage had facilitated the rehabilitation of the liver. Microscopic studies of the bile evidenced a rapid subsidence of the infectious process, for after 16 days of drainage no pus cells or bacteria could be found in the biliary secretions.

Biliary Dyssynergia: Obstruction of the Common Bile Duct by a Spasm of the Choledochal Sphincter.—There is a wealth of clinical evidence which indicates that a dyssynergia or spastic contraction of the common bile duct sphincter may produce an evanescent physiologic obstruction of the choledochal lumen and result in stasis of bile. Many surgeons have encountered a dilatation of the entire biliary tract but have been unable to demonstrate any pathologic impediment to account for the cholangiectasia. Krukenberg,[5] in 1903, reported a case of "gallstone colic" in which neither calculus nor infection was found. Aschoff and Basmeister[1] made an intensive study of the "stasis gallbladder" which occurred in the absence of stones, infections, strictures or anatomic deformities, and conceived the idea that a purely functional derangement of the choledochal sphincter might account for this condition. The work of Westphal,[14] Meltzer,[8] Lyon,[7] Berg,[2] Newman[10] and Ivy[4] indicates that a spasm or dyssynergia of the sphincter of Oddi may mechanically block the common duct and produce a stasis of bile with its attending colic and icterus. Newman, Nuboer,[11] and Giordano and Mann[3] have all reported cases in which a mild jaundice has resulted from a choledochal dyssynergia.

The diagnosis of biliary dyskinesia, or what we wish to call dyssynergia because of its more appropriate description, has previously been based on direct operative findings, supplemented by information obtained from duodenal intubation (Ivy, Newman and Meltzer) and the reaction of the biliary system to certain drugs (Westphal, Meltzer and Lyon). In our review of the literature we were unable to find roentgenographic evidence pointing to an actual spasm or dysfunction of the choledochal sphincter in man, and yet there is definite clinical and physiologic proof of its existence. Fortunately, while making these visualization studies of the bile ducts we encountered five patients who presented definite evidence of a dyssynergia or spasm of the common bile duct sphincter. Our findings were all corroborated by operative examination. The following case is representative of the group.

Case VI.—Mrs. A. C., housewife, aged 50, had suffered from paroxysms of nausea, vomiting and epigastric distress for the last one and one-half years. At times the sharp colicky pains became very severe. During the last nine months she had had a persistent jaundice, her stools had been acholic and she had lost 66 pounds in weight.

Examination revealed a poorly nourished, asthenic, deeply jaundiced woman. The liver extended two inches below the costal margin. The gallbladder could not be palpated. The stools were acholic but the urine contained bile. The van den Bergh reaction was positive in both the direct and indirect phases and the icteric index was 75. The general appearance of the patient, the rapid loss of weight and the persistent jaundice suggested either malignant changes in the hepatobiliary system or a pronounced liver damage from long standing calculous obstruction of the common bile duct.

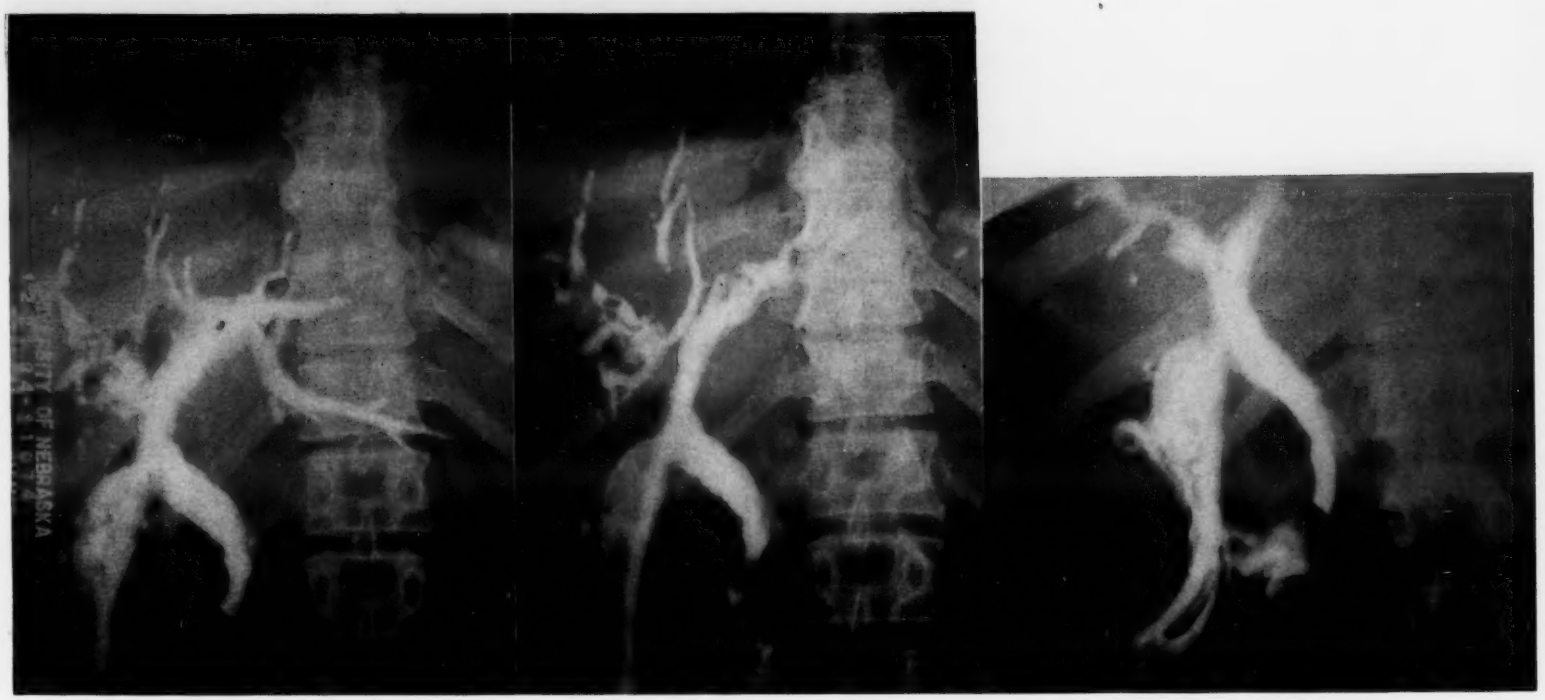

FIG. 9.—(Case VI.) On the fifth postoperative day 40 cc. of lipoiodine were injected into the common duct via its drainage tube. Note the pronounced dilatation of the bile ducts. The lipoiodine has encountered an obstruction at the distal end of the choledochus for the oil has not entered the duodenum.

FIG. 10.—(Case VI.) Ten minutes later. The choledochal obstruction is beginning to disappear as the oil is slowly trickling into the intestinal lumen.

FIG. 11.—(Case VI.) Thirty-five minutes after the injection. Note the stasis of lipoiodine in the larger bile ducts. The oil is flowing into the duodenum more rapidly. It required three hours before the obstruction had completely disappeared and the oil had passed into the lumen of the bowel. No obstruction was found at the operation. The location and the evanescent character of the obstruction signify a spasm of the choledochal sphincter.

After ten days of preoperative preparation, the abdomen was opened. The liver had a dark green, nutmeg appearance indicating a chronic retention of bile pigments. The dilated gallbladder contained many small stones and its walls were thickened, firm and indurated. The fundus of the gallbladder was angulated in such a manner that it was adherent to the common duct, there being a fistulous communication between the two. The common duct was about twice its regular size but no abnormalities could be found to account for this dilatation. Even after opening the choledochus, neither stones nor strictures could be felt; however, gentle irrigation with saline solution flushed out some fine gravel, several small calculi and inspissated plugs of bile-stained mucus. Catheters, probes and irrigating fluids were passed into the duodenum, thus attesting to the patency of the common duct. The gallbladder was held in firm apposition to the liver by dense adhesions and, because of the associated jaundice, it was felt unwise to attempt a complete cholecystectomy for fear of hemorrhage. The anterior and lateral walls of the gallbladder were excised, leaving the posterior portion intact in its hepatic fossa. The mucosa of the remaining gallbladder tissue was destroyed by carbolization and the muscular coats were firmly approximated by sutures. Catheters were placed in both the choledochus and common hepatic duct.

On the fifth postoperative day, 40 cc. of lipoiodine were injected into the common duct via the drainage tube. Roentgenograms which were made five minutes later demonstrated a pronounced dilatation of the larger biliary radicals, particularly of the right and left hepatic ducts. Interestingly the lipoiodine had encountered some impediment at the distal end of the common bile duct, for the oil had not entered the duodenum (Fig. 9). A second roentgenogram taken ten minutes later indicated that the obstruction was beginning to disappear as a small quantity of oil was then trickling into the intestinal canal (Fig. 10). Studies made 35 minutes later demonstrated a partial retention of lipoiodine. It required three hours for the obstructing mechanism to disappear so that the choledochus could evacuate its oily content into the duodenum (Fig. 11).

This case presented definite visual evidence of a temporary choledochal obstruction in spite of the fact that the operative examination revealed the common duct to be patent. At first, the blockade was complete (Fig. 9), but subsequent roentgenograms disclosed a gradual recession of the obstructive agent which finally permitted the oil to enter the intestinal lumen. The location of the mechanical block and its evanescent character indicated a spastic contraction of the choledochal-duodenal sphincteric mechanism. A spasm of this sphincter could readily occlude the orifice of the common duct and produce a dilation of the entire ductal system by causing a retention of bile.

CASE VII.—Mrs. J. W., housewife, 41 years of age, has had "gallbladder trouble" for the past five years. The colicky pains were associated with nausea, vomiting, passage of flatus and occasionally with jaundice. Two years ago a gallbladder filled with stones was removed and 22 pea sized calculi were extracted from the choledochus and hepatic ducts. Four months following the cholecystectomy she had a recurrent attack of colic associated with jaundice and several calcium bilirubin stones were recovered from the feces. Similar attacks were experienced during the next few months and on one occasion fragments of a large irregular stone were found in the stool. She began to lose weight, complained of weakness and had considerable abdominal distress. Intermittent chills followed by a temperature varying from 100° to 106° F. indicated a calculous obstruction of the common bile duct complicated by a cholangitis.

At operation, the choledochus was found to be as large as the duodenum and, on incising it, a thick yellowish pus escaped, indicating a suppurative cholangitis. A large

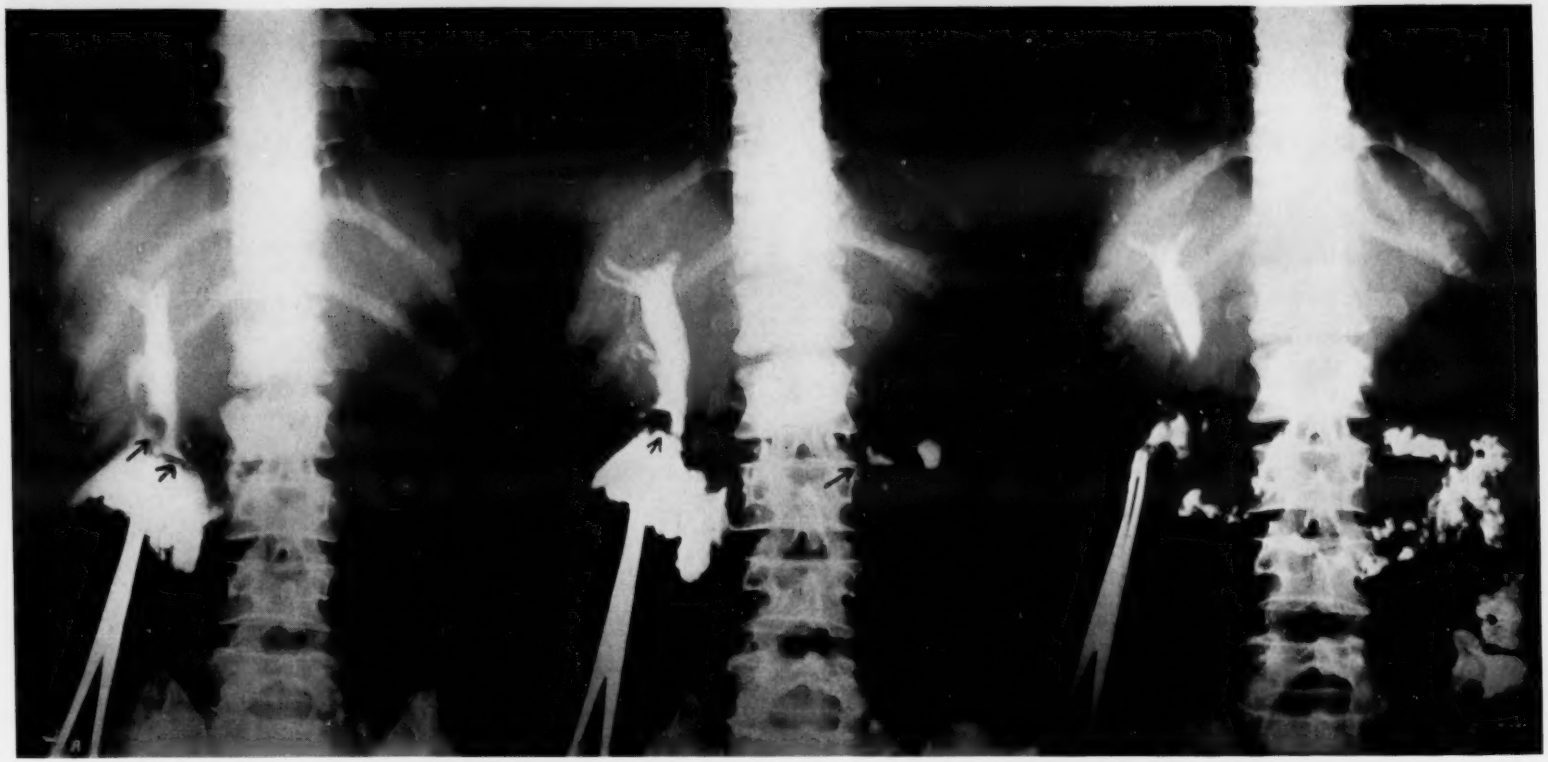

FIG. 12.—(Case VII.) On the sixtieth postoperative day 30 cc. of lipoiodine were injected into the external biliary fistula. The contrast media outlined a large subcutaneous cavity from which a small sinus tract led to the dilated common bile duct. At the terminal part of the choledochus there was a definite obstruction for the oil did not enter the duodenum.

FIG. 13.—(Case VII.) Progressive roentgenograms made three minutes later indicated that the obstructive mechanism was changing its form and some of the oil had escaped into the intestinal tract, but there was still a stasis of lipoiodine in the choledochus.

FIG. 14.—(Case VII.) Roentgenograms made 20 minutes later demonstrated that the obstructing mechanism had disappeared for the subcutaneous cavity and bile ducts had evacuated the oily content into the intestinal tract, signifying a relaxed and patent choledochal sphincter. Had the obstructing agent been a blood clot or stone, the blockade should have persisted rather than disappearing within 20 minutes' time.

calculus occluded the ampulla of Vater. This stone measured three-fourths of an inch in diameter and it was of soft mushy consistency. Careful investigation revealed the common duct to be patent so it was drained by a T-tube and a small catheter was placed in the dilated stump of the cystic duct.

On the first postoperative day, there was a slight subcutaneous hemorrhage which necessitated opening the wound and ligating the bleeding vessels. Five hundred cc. of yellowish mucopurulent bile were collected from the drainage tubes each day. On the tenth postoperative day the biliary discharge had a definite bloody tinge but the stools contained no blood. The bleeding became progressively worse in spite of repeated blood transfusions and the administration of several hemostatic substances. She began to complain of abdominal pain and it was apparent that intracholedochal bleeding had formed a blood clot which was obstructing the common duct as the amount of bile drainage through the tubes had practically stopped and her stools were acholic.

The abdomen was reopened on the fourteenth postoperative day and several blood clots were removed from the sinus tract. After removing the T-tube a large organized blood clot measuring 4 cm. in length and 1¼ cm. in diameter was extracted from the choledochus and hepatic ducts. A definite imprint of the T-tube was present on the anterior surface of the blood clot. There was a gush of bile from the hepatic ducts as soon as the obstruction had been relieved. The inner surface of the common duct was red, inflamed, and hyperemic, but the exact origin of the bleeding could not be determined. Instrumental examination demonstrated the ampulla to be patent, so a rubber catheter was sutured into the common duct. She was then given several postoperative blood transfusions.

Bleeding from the choledochus stopped spontaneously but there was considerable oozing from the abdominal wound and there was a profuse drainage of bile from the catheter. The stools were persistently acholic. She then developed a mass in the right lumbar gutter and a large fluctuant tumor in the pelvis. The culdesac was opened and six ounces of pus escaped. The hematoma of the right flank was drained.

During the next 21 days she gained in weight and the anemia improved; the biliary drainage was profuse, but her stools remained acholic. On a number of occasions the external biliary sinus was firmly packed with gauze but within two hours she experienced so much discomfort that the gauze plugs had to be removed. No bile was found in the feces even after the administration of atropine and magnesium sulphate. It seemed impossible to force the bile into the intestinal canal in spite of the fact that the choledochus was patent at operation. Hoping to determine the cause of this obstruction, a cholangiograph was made.

Sixty days following the first operation, 30 cc. of warm lipoiodine were injected into the common duct via the fistulous opening. The contrast medium revealed a large subcutaneous cavity communicating with enormously dilated bile ducts. There was a definite obstructive agent at the distal end of the common bile duct which prevented the lipoiodine from entering the duodenum (Fig. 12). A roentgenogram taken three minutes later demonstrated that a few drops of oil had escaped into the terminal duodenum, apparently indicating that the obstructive agent was beginning to disappear (Fig. 13). A third roentgenogram taken 20 minutes later revealed that the subcutaneous pocket and bile ducts had evacuated their contents into the intestinal tract, thus proving the choledochal sphincteric mechanism to be relaxed (Fig. 14). Immediately following the injection of the lipoiodine, the bile began to enter the bowel and within 48 hours' time the external biliary fistula had stopped draining.

It seems unreasonable to believe that a blood clot or residual stone could have been dislodged by the instillation of lipoiodine because she experienced no pain during this manipulation. It is to be remembered that the common duct was found to be patent at the operation, and yet no bile appeared in

the feces. Furthermore, the visualization studies indicate a gradual disappearance of the obstructive factor similar to that seen in patients having a choledochal sphincterismus. The most logical explanation of this unusual phenomenon is that the oil apparently produced a relaxation of the hyperspastic sphincter and established free drainage of the biliary passage.

Fig. 15 (Case VIII) represents an excellent cholangiogram of a patient who had a definite spasm of the choledochal sphincter, *i.e.*, biliary dyssynergia.

A functional dyssynergia of the choledochal-duodenal sphincteric mech-

FIG. 15.—(Case VIII.) This patient gave a typical history of biliary colic. At operation the gallbladder was found to contain numerous calculi and the bile ducts were all dilated. No gross obstruction of the common duct could be found. Forty-eight hours after the cholecystectomy had been performed, 40 cc. of lipoiodine were injected into the choledochostomy tube. Roentgenograms showed the choledochus and hepatic ducts to be dilated to three times their usual size. No stones, strictures or neoplasms were seen. In the terminal part of the common duct there was a mechanical obstruction which prevented the oil from entering the intestinal tract. Roentgenograms which were taken three hours later revealed that the obstructive agent had completely disappeared as the oil had escaped into the duodenum.

anism occurs much more frequently than we had thought. In a routine study of 11 patients undergoing operations for infections and calculi in the biliary system, four cases presented definite anatomic and roentgenographic evidence of this condition. Apparently the easiest and most accurate method of detecting the choledochal sphincterismus is by cholangiographic visualization.

What is the clinical significance of this biliary dyssynergia? A cardiospasm prevents the ingested foods from entering the stomach; a pylorospasm causes an obstructive gastric retention; a hypertonicity of the rectal

sphincter initiates constipation and fecal stasis; and a sphincterismus of the bladder neck interferes with micturition. It is not unreasonable, therefore, to believe that the choledochal sphincter can, by its spastic contraction, interfere with free flow of bile into the duodenum and thus produce a chronic biliary stasis.

If the accumulation and stagnation of bile within the ductal system is caused by the obstructive action of the hypertonic choledochal sphincter, then the removal of the gallbladder or drainage of the ducts will not correct the sphincterismus or ameliorate the distress of the patient. Repeated visualizations were made on such individuals for as long as 33 days after the operation and the dyssynergia still persisted.

Recognition of this choledochal dyssynergia by cholangiographic studies is very beneficial for corrective measures may then be employed. Berg has demonstrated that the sphincter is controlled by the autonomic nervous system; therefore, its spastic contractions can be overcome by using atropine. Newman and Ivy suggest that the duodenal instillation of olive oil and magnesium sulphate will relax the choledochal sphincter and drain the distended ducts. We have demonstrated on a number of occasions that lipoiodine injected into the common duct releases the sphincterismus or removes the offending obstruction. The use of such simple therapeutic procedures might mitigate the physical discomforts from which some cholecystectomized patients suffer. It is only by the direct visualizations that one can obtain graphic evidence of the functional status of the sphincter and when abnormalities are seen, then the proper therapeutic regimen can be instituted.

SUMMARY

(1) Cholangiography, or roentgenographic visualization of the gallbladder and bile ducts, is a very valuable diagnostic and therapeutic procedure. Diagnostically, it demonstrates the presence of calculi, strictures, neoplasms, fistulae, and attests to the patency of the bile ducts. It has a definite therapeutic value, for it helps to determine how long the dilated biliary system should be drained, it confirms the patency of the common bile duct before removing the drainage tubes, it demonstrates whether a cholecystostomy will decompress and drain the entire hepatic ductal system, it helps to ascertain the cause of an external biliary fistula, and occasionally a lipoiodine lavage of the common duct will flush out small stones or inspissated plugs of mucus which are causing distress and discomfort in cholecystectomized patients, or perhaps release the sphincterismus.

(2) Immediate cholangiography consists of injecting some radiopaque solution as lipoiodine directly into the gallbladder and bile ducts at the time of operation. Roentgenographic observations are made immediately following the injections of the oil and the operative procedures employed are determined by the radiographic findings. An example of the efficiency and simplicity of this method is given.

(3) Postoperative visualization studies of the extrahepatic ductal sys-

tem, or delayed cholangiography, are made by injecting lipoiodine into drainage tubes which are anchored in the gallbladder and bile ducts at the time of operation. These roentgenographic studies can be made at any desired time. The value of these observations is demonstrated by case presentations.

(4) Lipoiodine is a nontoxic, nonirritating substance which gives an excellent graphic reproduction of the entire biliary system. It was used in cases of acute cholecystitis, cholangitis, and in the presence of strictures, calculi, and pancreatitis without causing any local or systemic reactions.

(5) A spasm of the choledochal sphincteric mechanism or biliary dyssynergia is discussed and roentgenographic demonstrations of its existence are evidenced by delayed cholangiography.

(6) A cholecystostomy, to be of the most value, must not only drain the gallbladder but it must decompress and drain the entire hepatic ductal system. If the cystic duct is occluded, then only the gallbladder will be drained and the bile ducts may still retain the stagnant bile and the intraductal pressure will not be reduced. Cholangiography is very valuable in determining the patency of the cystic duct.

(7) The beneficial effects of prolonged drainage of the bile ducts in cases of cholangiectasia and cholangitis are discussed and cholangiographic observations are presented.

(8) The introduction of lipoiodine into an external biliary fistula gives valuable information concerning its etiology as well as suggesting the most feasible method of correction.

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DIVERTICULOSIS OF THE SMALL INTESTINE

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THE vast majority of acquired diverticula of the small intestine are of the mucous membrane hernia type, similar to the pouches so frequently to be found in the large intestine.

The diverticula are usually multiple, and are practically limited to the upper part of the jejunum, the ileum being affected only in extreme cases.

Historic Note.—The first complete description to be found in the literature of multiple jejunal diverticula, in which no doubt exists as to the nature of the diverticula in question, was by Sir Astley Cooper[1] in 1844, who found numerous pouches of the upper part of the jejunum in a man of 65. The condition appears to have excited little comment at that time, and very few other references are to be found until the last decade of the nineteenth century, though Osler[25] in 1881 found at postmortem in a man of 65, who had suffered from rumbling noises after food, 55 diverticula in the upper part of the jejunum, ranging in size from a cherry to a large apple.

In the following decade, Virchow,[36] who described a case of "Hernias in the Jejunum and Ileum," Edel, Grassberger,[13] Henseln, Hansemann,[16] Hanau,[15] and Nicholls,[24] all describe personal cases, and give their views of the pathogenesis. Hansemann's case is the most famous. He found no less than 400 diverticula in a single case—a man of 85. Some of these were duodenal, and some in the upper part of the ileum, but the vast majority were jejunal.

Keith included a description of a specimen of jejunal diverticula in an article written in 1910, and there have since followed descriptions of the condition by Taylor and Lakin[33] (1910), Latarjet and Murard[20] (1914), Terry and Mugler,[35] and Mackechnie (1921), Caraven and Helvestine[14] (1923), Berry[4] (1927), Lund (1928), Jenkinson,[17] and Weiss (1929), Boling[5] (1930), and Tengwall[34] (1931). In 1922, Braithwaite[6] published his remarkable operative case, to which the writer, who has had the opportunity to examine the specimen, will subsequently refer.

Material.—The following description of the morbid anatomy and pathogenesis of acquired diverticula of the jejunum and ileum is based upon an examination of six postmortem and three operative specimens.

Unlike duodenal diverticula, pouches lower down the small intestine are difficult to detect by roentgenography, except in the comparatively rare cases in which there is retention of barium in them, and they will, therefore, be more often missed than not.

In 4,631 barium meals the author found only four cases in which

diverticula could be diagnosed with certainty. This makes the percentage incidence 0.086 per cent, a figure probably far too low. Jenkinson[17] records three cases in many thousand roentgenograms.

Autopsy Statistics.—2,820 autopsies with nine cases of diverticula (0.31 per cent). This is a more reliable guide than roentgenography, but is still too low. During the past four years, in which 881 autopsies have been made, and in which particular attention was given to the search for diverticula, five cases, either single or multiple, have been discovered, an incidence of 0.57 per cent. In these cases the criterion of the acquired nature of the diverticulum has been absence or deficiency of the muscular coat. This is probably a more accurate figure of incidence, and agrees more closely with the expressed opinions of many postmortem workers.

The author's seven postmortem and three operative cases are summarized in Table I.

TABLE I

Case	Situation	Number	Sex	Age	Clinical Notes
1 (Fig. 1)	Upper part of jejunum	18	M.	55	Death from pulmonary embolism. No symptoms noted during life
2 (Fig. 5)	Upper jejunum	6–8	F.	38	Operative specimen
3	Upper part of jejunum	6	F.	63	Road accident; death No history
4 (Fig. 6)	Jejunum 18 ins. from flexure	1	M.	48	Death from cerebral thrombosis. No symptoms noted during life
5 (Fig. 7)	Jejunum	1	M.	7	Native of Madras. No clinical history
6 (Fig. 8)	Jejunum	1	F.	62	Death from cerebral tumor
7 (Fig. 11)	Jejunum	1	F.	57	Malignant growth affecting jejunum
8 (Fig. 17)	Ileum or lower end of jejunum	1	M.	53	Operative case. Acute inflammation of the diverticulum
9	Jejunum	1	M.	49	Operation. Diagnosed by roentgenogram. Diverticulum excised
10 (Fig. 10)	Jejunum 9 ins. from flexure	1	M.	55	Death following partial gastrectomy for gastric ulcer

Eight of these cases have been examined histologically and they are all of the acquired type, with the probable exception of No. 8. In all but one of the cases the pouch or pouches arise from the mesenteric side of the intestine. No. 6 is the exception, the single diverticulum lying opposite the mesenteric attachment.

In one of the cases a malignant growth was associated with the pouch. This was an unique case, and, though it is impossible to establish a cause and effect formula with regard to the pouch and the growth, the case must be classified apart from the others.

To these must be added the remaining two of the four cases diagnosed roentgenographically, in whom operation was not performed (Table II).

TABLE II

Case	Situation	Number	Sex	Age	Clinical Notes
11	Jejunum: upper part	Multiple	M.	67	Flatulent dyspepsia
12	Jejunum: upper part	Multiple	M.	69	Flatulent dyspepsia

Age and Sex Incidence.—The age incidence is a relatively high one, the youngest patient being 49, the oldest 69. The average age for 11 cases was 56 years. Eight were men and four were women.

In the 15 cases of multiple diverticula which the author has selected from the literature as being undoubted examples of the type of diverticula under discussion, the average age in 14 was 62, the youngest 43, and the oldest 85 years. The latter case contained 400 diverticula. Of the 15 cases nine were men and six women (Table III).

TABLE III

	Sex	Age	Number
Cooper	M.	50	Multiple
Osler	M.	65	53
Virchow	M.	old	Multiple
Good	F.	77	6
Hansemann	M.	85	400
Nicholls	F.	64	45
Gordinier and Sampson	F.	46	13
Taylor and Lakin	F.	68	Multiple
Braithwaite	M.	45	60
Case	M.	61	12
Case	M.	73	25
McWilliams	M.	71	7
Terry and Mugler	F.	59	5
Mackechnie	F.	43	13
Helvestine	M.	63	58

It should be borne in mind that the mean age calculated from these figures represents the age of discovery of the diverticula. The mean age of development would be a much lower figure.

PATHOLOGIC DESCRIPTION OF THE AUTHOR'S CASES

SPECIMEN I.—The specimen (Fig. 1) was obtained at postmortem upon a man of 55, who died from a pulmonary embolism following an operation upon the bladder. There had been no indication of the presence of the diverticula during life.

The specimen consists of the upper part of the jejunum, commencing two inches below the duodenojejunal flexure. This segment of intestine is studded with diverticula varying in size from that of a split pea to that of a chestnut. In all, 18 can be counted. The diverticula are all situated at or near the mesenteric border of the intestine, pushing their way between the leaves of the mesentery. Looking at the specimen from the lateral aspect, a well defined vessel is seen running in the thin wall of each pouch and continuing, but less clearly seen, on the wall of the bowel towards the antimesenteric edge. These vessels are evenly spaced, and the diverticula, though varying in size, are also equally spaced, each of them being related to a segmental vessel.

If the specimen be viewed now from the mesenteric side, it is seen that the largest diverticula bestride the mesenteric angle, pushing aside the leaves of the mesentery. The opening of the pouches into the bowel is a wide one, and is placed symmetrically over the line of normal mesenteric attachment. The smallest diverticula, however, lie to one side of the mesentery, and their opening into the bowel is lateral to the normal line of mesenteric attachment. Each of the small diverticula is paired. Its fellow is situated equidistantly from the mesenteric line, and on the opposite side. The diverticula of each pair are unequal in size.

The pouches intermediate in size between the largest and the small paired ones are bilobed. The mouth of such a diverticulum bestrides the mesenteric line, and the leaves of the mesentery are reflected away from the intestinal wall as in the case of their larger neighbors. At the fundus, however, there is a grooved depression in the long axis of the diverticulum corresponding with the line of attachment of the mesentery to the normal intestine, and giving each diverticulum its bilobular character. This appearance is represented semidiagrammatically in Fig. 2.

FIG. 1.—Diverticulosis of the jejunum. Postmortem specimen. The pouches are all closely related to the mesentery.

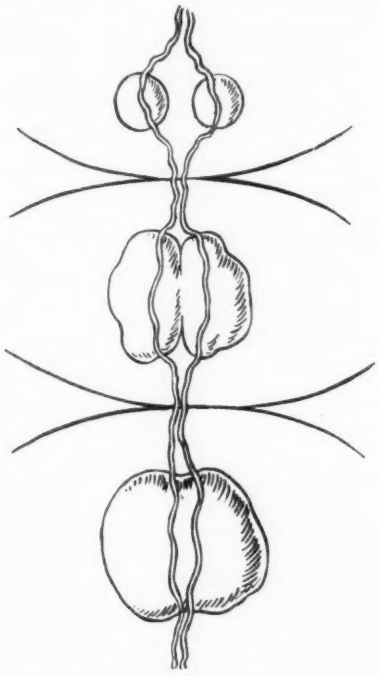

FIG. 2.—Semidiagrammatic drawing of a portion of the specimen illustrated in Fig. 1, viewed from the mesenteric aspect, showing the mode of formation of the pouches.

Figure 3 shows a portion of the specimen opened up by cutting along the exact line of the original mesenteric attachment. Valvulae conniventes are present as in the normal intestine.

The separate openings of the small paired diverticula are now clearly shown, one on either side of the mesenteric line. The lower two pairs show the diverticula to be of unequal size, as though one had started life before its opposite, and that from the chronological standpoint the two are not twins. The mouth of each is sheltered by a valvula connivens.

In the third segment of the bowel (from below upwards) the mouth of a medium sized diverticulum has been cut through by the incision. The greater portion of the mouth is on the left side as one looks at the picture (Fig. 3).

The mode of formation of each large diverticulum is clear. It started originally as a pair of pouches, one on either side of the line of the mesentery. Later in development the pair became larger, until they met in the line of the mesentery, and the thin strip

FIG. 3.—The specimen illustrated in Fig. 1 opened by cutting along the line of attachment of the mesentery. Note that the smaller diverticula are paired, each opening immediately lateral to the mesenteric line.

of bowel which separated them originally, became taken up, and the mouths of the two pouches fused. At this stage, the fusion of the pouches was indicated externally by the median longitudinal groove seen on the external aspect on the line of the mesentery. Eventually, as a result of continued pressure from within the bowel, this median groove became obliterated, and a single smooth-walled large diverticulum resulted.

The point which is established is, therefore, that the site of origin of each diverticulum is not at the line of mesenteric attachment to the bowel, as the appearance of the fully developed pouches suggests, but to one or other side of this line. This situation corresponds exactly with the entrance of the blood vessels through the muscular coat of the intestine, as will be described in the succeeding pages.

If the opened specimen is held up to the light, it is at once evident that the walls of the pouches are thinner than the wall of the neighboring bowel. Each diverticulum is thinnest at its fundus.

Figure 4a is a low power (7×) photomicrograph of a section taken through one of the small diverticula. The diverticulum opens by a wide mouth into the parent bowel, and is hemispherical in shape. The mouth is bounded on the right side as one looks at the illustration by a valvula connivens. At the fundus of the pouch are a large vein and artery, and smaller branches of these vessels can be seen in both of the angles between the diverticulum and the jejunum.

The mucous membrane lining the diverticulum shows no difference in detail from that of the bowel proper, but as this is a postmortem specimen, the mucous membrane is poorly preserved. There are no rugae present in the diverticulum comparable with the valvulae conniventes of the normal bowel.

The wall of the diverticulum is thinner than that of the intestine, and this is due entirely to the difference in width of the respective muscular coats. The fibers of the intestinal muscularis appear to be heaped up at the angles between the pouch and the intestine. On placing a finger in the diverticulum this is felt as a firm prominent edge

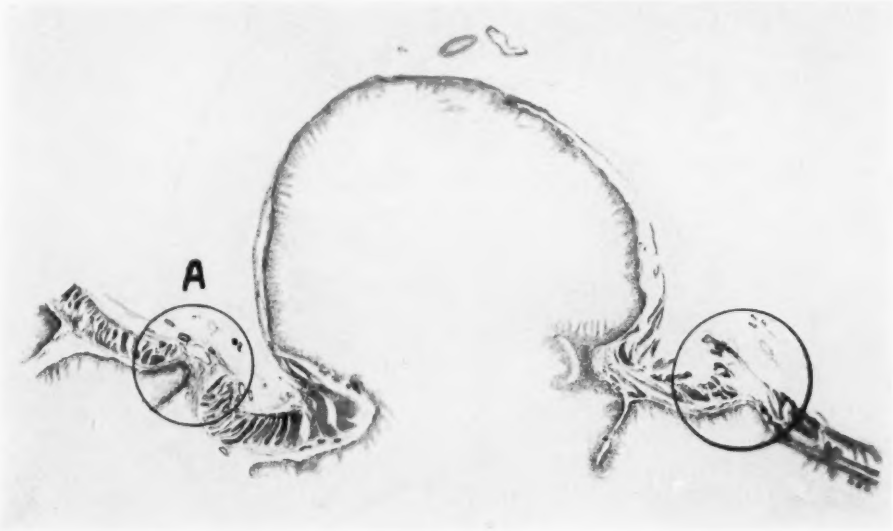

FIG. 4a.—Photomicrograph of a section through one of the diverticula (×7). Note the vessels at the fundus of the pouch and the absence of a muscular coat.

surrounding the diverticulum, almost like a sphincter. At the commencement of the diverticulum, looking at the left side of the illustration, the muscular coat is pushed back upon itself, and is then continued into the diverticulum as a thin layer not more than two to three fibers deep. The layer completely surrounds the diverticulum, getting slightly thinner still as it approaches the intestine on the right side of the mouth. There is a slight interruption in the muscular layer here—probably an artefact.

The muscularis mucosae can be seen on high power examination as a thin layer of small fibers, continuing in an unbroken line from the intestine, underneath the mucous membrane of the diverticulum. These appearances strongly suggest that the mucous membrane has been pushed through a weak area in the muscularis of the bowel carrying with it a thin layer of muscle fibers—a pulsion diverticulum. The point at which this occurred in the intestinal wall corresponds with the entrance of the large blood vessels seen at the fundus of the diverticulum.

At either side of the mouth of the diverticulum, and identified by inclusion within circles, are two areas in which there is a gap in the musculature occupied by blood vessels. A pouching of the mucous membrane into these gaps is seen. The gap on the

left side is better shown, and Fig. 4b is a higher power illustration of this field. Here can be seen a wide interval in the well developed muscularis of the jejunum, occupied by a number of blood vessels. The largest of these is a thin-walled vein, and this corresponds with the apex of a pouch of mucous membrane, which is herniating through the vascular interval to form a diverticulum. From this illustration, one can readily appreciate how the contraction of the muscle at the mouth of the pocket of mucous membrane will force it further into the gap occupied by the vessels and how increased pressure within the lumen of the jejunum will foster its development into a complete hernial diverticulum. As the diverticulum passes through the muscular coat to form a protrusion on the peritoneal aspect of the intestine, it will probably take with it muscular fibers from the wall of the bowel, which will furnish it with a thin muscular coat. At the fundus of the diverticulum, however, there will clearly be no muscle fibers at all, except those belonging to the muscularis mucosae.

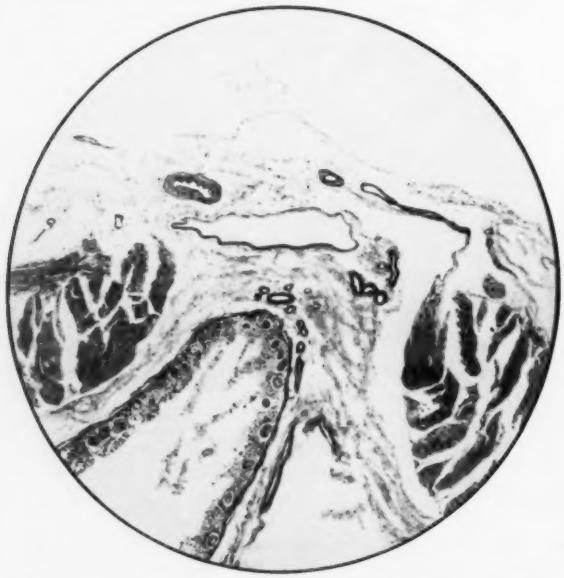

FIG. 4b.—Photomicrograph of higher magnification of area enclosed within circle A, Fig. 4a. Note the vascular gap in the muscularis partly occupied by a pouch of mucous membrane.

SPECIMEN II.—The specimen illustrated in Fig. 5 was removed at operation from a woman of 38 who had, for upwards of eight years, complained of flatulence and discomfort, sometimes pain, after food. Six to eight pouches are present and their morphology is *identical* with those of Specimen I. There is a nodule of aberrant pancreatic tissue on the antimesenteric aspect of the bowel.

SPECIMEN III.—The third case in the series was sacrificed in order to obtain microscopic sections through all of the six diverticula present. The diverticula were spaced out along the mesenteric border at regular intervals of about five inches. Each pouch was related to a large blood vessel at its entry through the wall of the intestine. Four of the pouches were single, but two were situated next one another on opposite sides of the mesenteric angle. None of the pouches contained any muscle at the fundus with the exception of small thin fibers belonging to the muscularis mucosae.

SPECIMEN IV.—In this case there is a solitary diverticulum situated 18 inches from the duodenojejunal flexure (Fig. 6). It was obtained postmortem from a man of 48, who died from cerebral thrombosis. No symptoms referable to the presence of the diverticulum were noted during life.

The diverticulum is the size of a small chestnut. It is situated wholly to one side of the mesentery, and does not push between the leaves. There is no trace of any

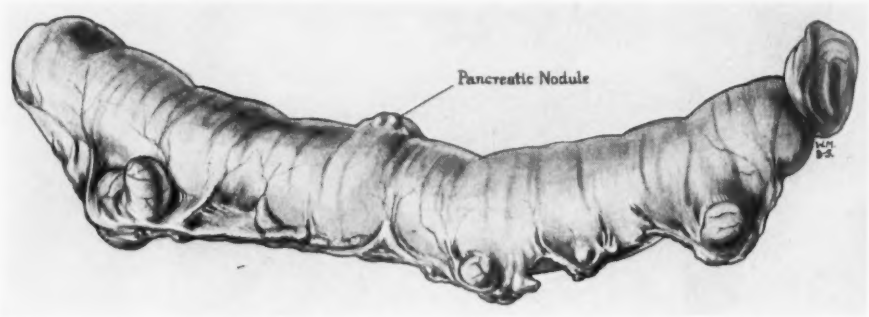

FIG. 5.—Diverticulosis of the jejunum. Operative specimen. There is an aberrant pancreatic nodule on the antimesenteric aspect of the bowel.

diverticulum on the reverse side of the mesentery on gross examination, though the possibility that an early one is present and would be revealed by microscopic section cannot be denied when one considers the mode of formation of the largest diverticulum described in Specimen I. There is a vessel associated with the diverticulum, and branches of this can be seen in the lateral view under the peritoneal coat.

Microscopic section of the jejunal wall shows nothing abnormal, beyond a poorly staining mucous membrane, due to postmortem digestion. A section of the diverticulum passing through the angle formed by it and the intestine is illustrated in Fig. 7. The muscularis of the latter is doubled back upon itself so that it appears thicker than normal and looks almost like a sphincter. As the muscle coat passes into the wall of the diverticulum, it rapidly becomes thinned. The longitudinal fibers suffer first, but both layers share in the process, and at a distance of 0.5 cm. from the mouth of the diverticulum all that remains of the muscularis is a thin sheet of muscle, not more than 2 to 3 fibers in breadth, which by the direction of their long axis appear to be remnants of the circular layer.

The submucosa has practically disappeared in this specimen, and the mucous membrane is a little thinner than in the bowel. Two or three vessels are present in the angle, lying underneath the peritoneal coat, and standing some distance from the wall of the bowel (an artefact).

FIG. 6.—Solitary diverticulum of the jejunum, situated immediately lateral to the mesentery.

SPECIMEN V.—This specimen (Fig. 8) was sent to the author by a doctor practicing in Madras, who stated in his letter that such diverticula were a common finding at postmortem among the natives of that part of India.

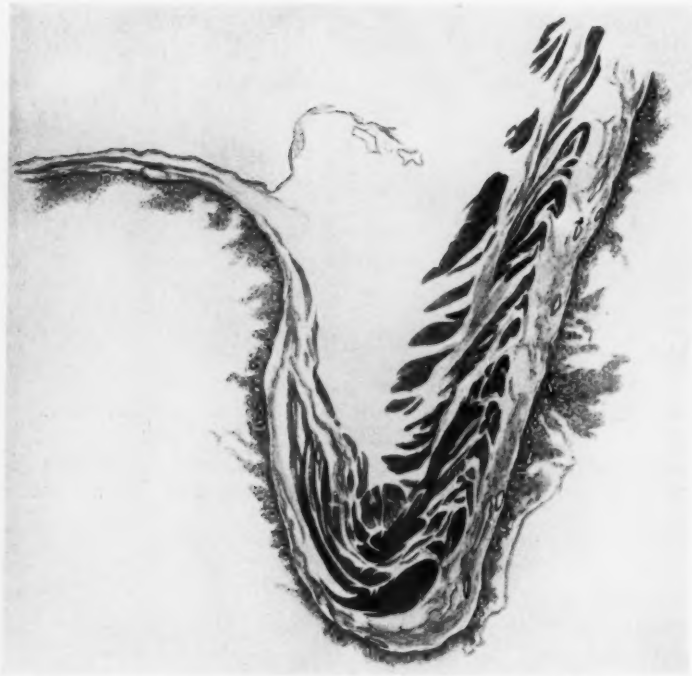

FIG. 7.—Photomicrograph of a section of the specimen illustrated in Fig. 6 (×7). Note the heaping of the muscular coat in the angle.

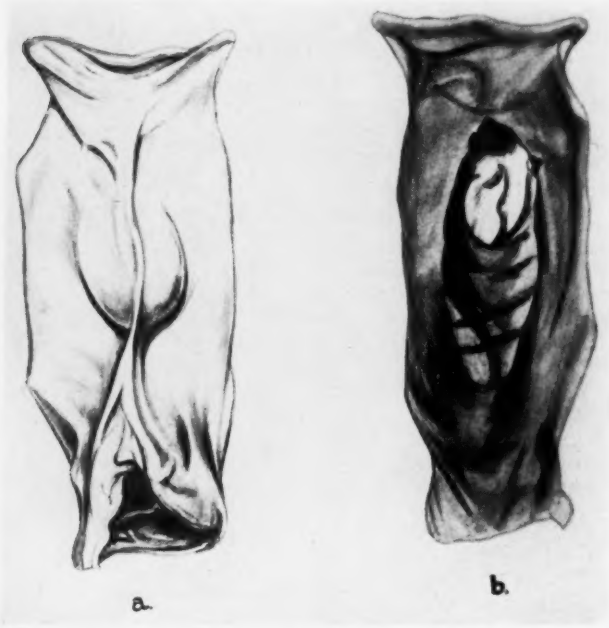

FIG. 8.—(*a*) Solitary jejunal diverticulum. It bestrides the mesenteric angle; (*b*) was drawn with the specimen hung in front of a bright light, and demonstrates the thinness of the wall of the pouch.

There is a single diverticulum pushing its way into the leaves of the mesentery, somewhat flask shaped, and opening into the intestine by a wide mouth. The mesentery is not placed symmetrically across the diverticulum, but divides into two unequal portions, suggesting that the diverticulum is developed by fusion of two diverticula of unequal size. Viewed from inside the lumen of the bowel, the mouth on one aspect is bounded by a sharp crescentic ridge. On the other aspect the lumen of the gut slopes down into the diverticulum without such interruption.

The illustration Fig. 8b was made with the specimen hanging down in front of a bright light. The diverticulum throws a shadow to the left side of the mesentery. The greater translucency of the wall of the diverticulum as compared with that of the normal bowel is clearly shown.

The miscroscopic section made of the wall of the diverticulum shows an absence of true muscle coat, the latter ceasing abruptly at the mouth of the pouch.

SPECIMEN VI.—This specimen (Fig. 9) was obtained at postmortem upon a woman of 62, who died from a cerebral tumor.

There is a small diverticulum on the *antimesenteric* border of the jejunum, and in view of its situation the case is unique. It is not a Meckel's or any other type of congenital diverticulum as microscopic examination proves, but a pulsion or primary acquired diverticulum. Running into its wall is an artery of unusual size—much larger than any of its collaterals. Instead of breaking up into terminal branches half way along the wall of the intestine, as do its neighbors, this vessel pursues an uninterrupted course along the normal intestinal wall right on to the wall of the diverticulum, where it finally terminates in small branches.

The diverticulum is not flask shaped like the other specimens described, but its mouth is equal in width to its largest diameter.

SPECIMEN VIII.—The diverticulum opens from a portion of the small intestine resected at operation because of acute inflammation of its wall. There is some doubt as to whether it is ileal or jejunal. The case will be described in detail subsequently.

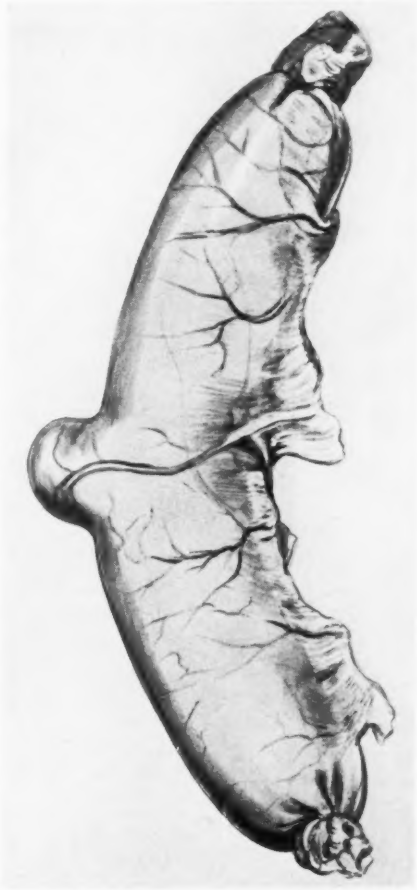

FIG. 9.—Solitary diverticulum of the jejunum. The specimen is unusual in that it arises from the antimesenteric aspect of the bowel. Note the large vessel associated with it.

SPECIMEN X.—This was diagnosed roentgenologically (Fig. 10), and its presence confirmed at operation. It opened from the mesenteric aspect of the jejunum nine inches from the flexure. The wall was thinner than the normal intestinal wall, but no microscopic examination was made.

SPECIMEN VII.—This is unique, in that it is associated with a malignant growth of the bowel (Fig. 11). The diverticulum is situated on the mesenteric aspect of the jejunum, to the side of the mesentery, and possesses no muscle coat.

SUMMARY OF THE MORBID ANATOMY

(1) Of a total of 12 cases from all sources, five are multiple and seven single.

(2) Specimens VII and VIII present unusual features, and will be considered separately.

(3) The diverticula of Specimens I, II, III, IV, V, VI and IX, and probably X, are herniae of the mucous membrane of the jejunum. There is no difference in morphology between the single and the multiple cases.

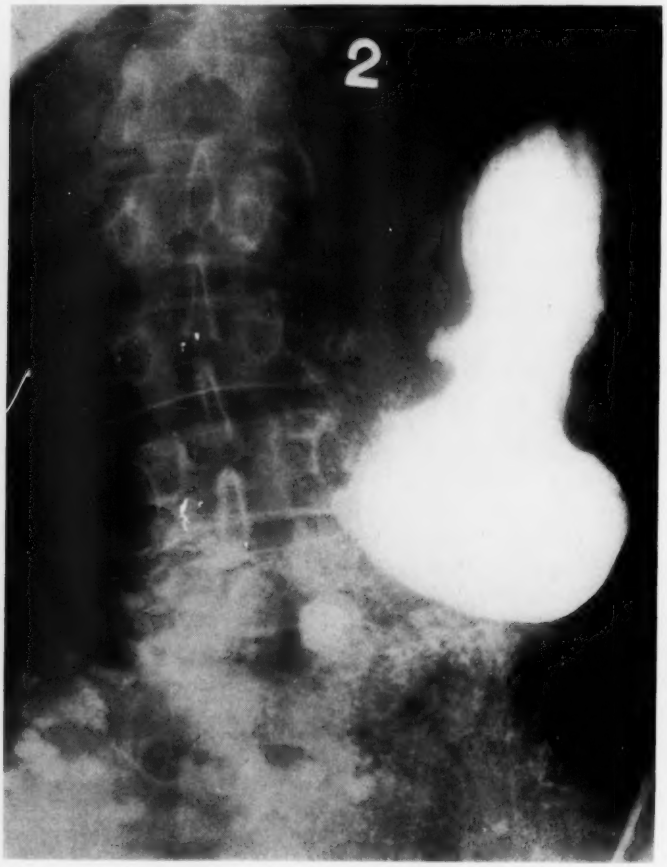

FIG. 10.—Solitary diverticulum of the jejunum associated with a chronic gastric ulcer (position of pouch confirmed by inspection).

(4) The site of herniation of the mucous membrane through the wall of the intestine corresponds with the entry of the blood vessels.

(5) In all except one case, the diverticula are on the mesenteric aspect of the intestine. They do not, however, push between the leaves of the mesentery unless they are of large size, or fuse with a fellow from the opposite side of the mesentery. In the case of the diverticulum on the antimesenteric side of the intestine, the opening corresponds with a blood vessel of unusual size.

(6) The diverticula may carry with them during their formation a layer of muscle fibers from the muscular coat of the bowel. Such may form a thin but complete muscular coat for diverticula of small size, though it seems probable that if a section were examined which chanced to pass through the point on the fundus of the diverticulum which was the first to pass through the muscular coat of the bowel, no muscle fibers would be seen. In large diverticula, the fundus is completely devoid of a muscular coat. This is because it increases in size chiefly at the expense of the mucous membrane and submucosa, and eventually there is not sufficient muscular tissue in its

FIG. 11.—Solitary diverticulum of the jejunum associated with a new growth.

wall to "go round." The latter is limited, therefore, to the walls at the commencement of the diverticulum.

Pathogenesis.—There is no room for doubt that the diverticula under discussion are acquired deformities of the bowel wall. Apart from the age incidence—diverticula of this type are unknown in the new born—their pathologic anatomy is to be explained only by the thesis that the pouches are herniae of the mucous membrane through the muscular wall of the intestine. If this view be accepted, the causal factors to be discussed are two.

(I) The presence of a weakened area—a locus minoris resistentiae—in the bowel wall.

(II) A pulsion force acting from within the bowel which initiates the process of herniation.

I. The Locus Minoris Resistentiae

The constancy with which these diverticula are associated with the blood vessels refutes the possibility of coincidence. Furthermore, it has been shown in the analysis of Specimen I that the diverticula start just to one or the other side of the mesentery. As the following anatomic description shows, this coincides with the perforation of the muscularis by the blood vessels supplying the intestine.

The Anatomy and Rôle of the Blood Vessels.—Fig. 12 is an illustration

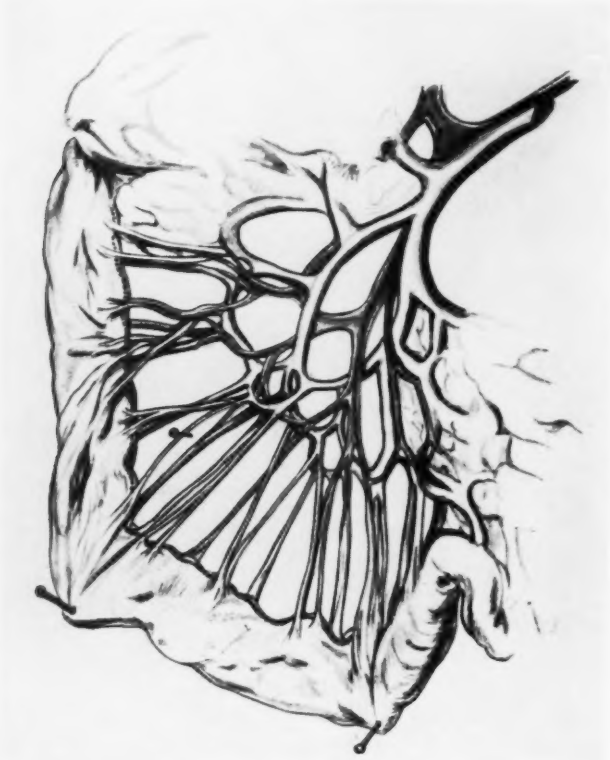

Fig. 12.—Dissection of the vascular supply of a segment of the jejunum. The midline is indicated by the interrupted line. The terminal vessels enter the bowel on either side of the midline.

made from a dissection of a segment of an adult jejunum, after injecting the superior mesenteric artery *in situ* in the dead body.

The mesentery and mesenteric fat have been cleaned off the vessels, and the artist has faithfully represented the extremely complex interrelation of the arteries and veins. One inch or more from the margin of the intestine the final series of arches is formed, and from these arches the segmental arteries pass to the intestinal wall.

The arteries pierce the intestinal wall in pairs and on either side of the mesenteric line, which is shown by the interrupted line in the illustration,

and a variable distance from it, but not more than three-sixteenths of an inch away.

In most cases the arteries of each pair are exactly opposite each other, but in some cases they alternate. There is, in other words, not a completely regular arrangement. When the arteries reach the wall of the intestine, on

FIG. 13.—Photomicrograph of a whole section of jejunum after injection of vessels with carmine jelly. Note how the artery and vein interrupt the muscularis just lateral to the midline.

either side of the mesenteric line, they disappear from view by piercing the muscular coat to reach the submucosa. Some fine twigs, omitted for the sake of clearness from the illustration, are given off before the main vessels pierce the muscular coat, and pass towards the antimesenteric border between the muscular and serous coats.

A cross section of an adjacent portion of the jejunum was now made to show the way in which the muscular coat is pierced by the blood vessels. Fig. 13 illustrates a low power photomicrographic section of this. The section passes through the long axis of one of the blood vessels as it pierces the muscular coat.

It will be noted that the blood vessel passes through both muscular coats a little distance from the mesenteric line, producing a well defined gap. The entrance is at about 60° to the muscular coat so that the gap is not a very oblique one. *Practically the whole* of the blood supply to one-half of a segment of intestine is conveyed by this single vessel, which terminates in branches in the submucous coat. Some of these small branches are shown at the antimesenteric aspect of the bowel. They are somewhat distended as the result of injection under pressure. There are thus only two large gaps occasioned by the entry of the blood vessels of the jejunum in each segment, and those gaps are situated on either side of the mesentery, and a short distance from it (Fig. 14). The angle of penetration of the vessels, moreover, is such that the opening in the longitudinal coat is directly in line with that in the circular coat. Where any branches pierce the muscular wall farther away from the mesentery, not only are the gaps much smaller as the vessels are smaller, but the angle of penetration is far more oblique, and thus does not tend to create the same degree of weakness in the intestinal wall. The vessels of each segment are situated in most instances opposite one another, but in others they alternate.

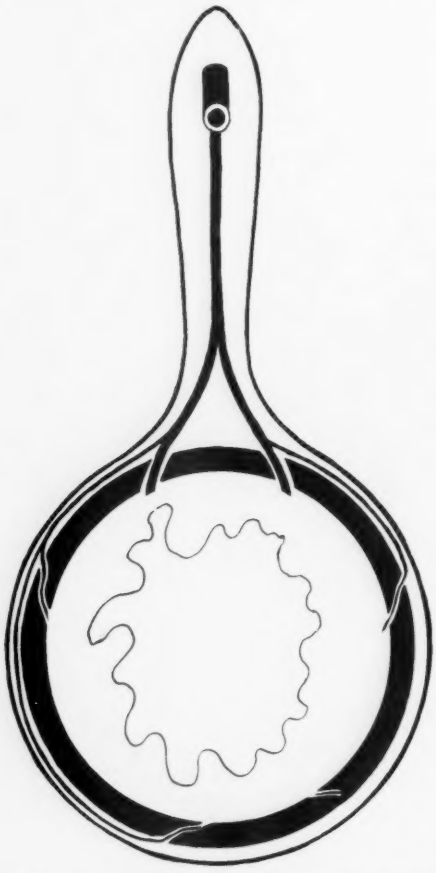

FIG. 14.—Diagrammatic representation of the mode of entry of the blood vessels through the muscular wall of the bowel.

Thus we see that the origin of jejunal diverticula corresponds exactly with the point of entry of the blood vessels through the muscular coat. In only one instance in this series is this rule broken (Specimen VIII, Fig. 9), and in this there is an abnormality in the vascular supply, a very large vessel traversing the intestinal wall between the peritoneal and muscular coats and probably passing through the latter near the antimesenteric border, and producing a gap in the latter coat at this situation.

Thus the evidence that these diverticula are herniae of the mucous membrane through the muscular wall, at the point where a gap is caused in the latter by the entrance of the blood vessels, is indisputable.

One cannot leave this aspect of the subject without some reference to the work of Fischer. In general the author's conclusions are similar to Fischer's but the latter has made one or two additional observations from his own examination of the morbid anatomy of jejunal diverticula. He states that the circular muscle is the first to go and the outer coat remains intact for some time, but eventually becomes thinned as the diverticulum gets larger, and eventually disappears. This order, however, is not invariable. On occasion, the mucous membrane and inner circular layer pass through the longitudinal coat, and sometimes the break occurs in both circular and longitudinal cases simultaneously. Fischer does not think the diverticula always follow the blood vessel. Once started through the gap caused by a blood vessel, a pouch may track along the line of least resistance. He also states that in some cases there is thickening of the peritoneal covering of the diverticulum, which he interprets as nature's attempt to limit it.

In my mind it is questionable whether the blood vessels play any further part in the pathogenesis other than that of providing the locus minoris resistentiae. Klebs,[18] however, believed that the mesentery increased in breadth in relation to the arteries, and therefore the latter dragged upon the submucous coat at their points of entry into the bowel, tending to pull it up through the muscularis. One frequently sees "festooning," as Craven has described it, of the small intestine wall at operation and postmortem. It may be due to the pull of the blood vessels, and it is a possible predisposing cause of herniation. It is unlikely, however, to play a major part in the process.

Sudsuki[32] suggested in 1900 that the weak areas in the bowel wall are due to degeneration of the connective tissue sheath of the veins, so that an interval is created between the veins and the edges of the gap they caused in traversing the muscle coat. The theory is entirely speculative, and has no counterpart elsewhere in the body. Sudsuki appears to have offered this theory purely to replace that of Graser.[12] The latter suggested that the weak area in the muscular coat was produced by congestion of the veins draining the intestine, basing his conclusions chiefly on the finding that, in a postmortem series of pulsion diverticula of the large intestine, a number of the subjects suffered from chronic cardiac disease.

Perivascular infiltration of the muscle with fat has been suggested as a cause of the weak areas, but the author has found no suggestion of such a process in the specimens under review.

Some observers have suggested a myogenic cause, such as fatty degeneration (Roth), congenital hypoplasia (Buzzi[9]), senile atrophy (many workers), and a toxic myositis (Spriggs and Marxer[31]). The possibility that such degenerative processes are a cause is ruled out by the constancy with which these diverticula are situated in relation to the blood vessels.

II. The Pulsion Force

We are ignorant of the variation in pressure that must occur within the jejunum and ileum during life. Experimental evidence in animals obtained by placing balloons in a segment of the jejunum isolated by operation, and similar experiments upon the subjects of artificial jejunostomy, must be discounted for the following two reasons:

(1) The conditions are abnormal in that a stoma is present.
(2) The presence of an inflated balloon will provoke contractions in the musculature differing from any that are likely to arise spontaneously. Most of the results of this type of experiment record only the passive pressure possible to obtain by distension from within the intestine, and do not register the pressure excited by active contractions of the jejunal musculature under normal conditions.

The time honored name of jejunum is derived from the emptiness of this section of the intestinal tract found at postmortem. At operation the jejunum is collapsed and apparently empty, with a rugose and flaccid wall unless there be organic obstruction. Under normal conditions of segmental contraction and peristaltic movement the increase in pressure must be momentary and insignificant, for there is no bar at either end to the passage of the contents, such as the pyloris may furnish to the duodenum.

Can the passive force of the contents of the jejunum create a pressure sufficient to cause herniation? All writers appear to accept the view, first put forward by Klebs[18] in 1896, that the pressure of fluid and gas in the jejunum is the initiating force of the diverticula. No intrajejunal tension under ordinary conditions can approach in power that present in acute small intestine obstruction. The tense, ballooned jejunum of organic obstruction is all too familiar to surgeons, but the condition never gives rise to hernial diverticula. The production of diverticula of the intestine by distending the dead bowel under pressure has frequently been attempted.

Thus Hanau[15] filled a loop of intestine with water and by subjecting it to hydraulic pressure produced trench-like furrows along the mesenteric border before perforation occurred. Hansemann,[16] by the same method, claims to have produced diverticula related to the blood vessels. These were obtained only when using intestines from old people. He failed with the intestines of children.

It cannot be too strongly emphasized, however, that experiments upon the dead intestine are entirely without value. The strength of the intestinal wall depends largely upon the strength of its muscular coat, and the essence of the strength of muscle lies in its power to contract.

Chlumsky has shown by experiment the fallacy of drawing conclusions from any distension experiments upon dead intestine. He found that by distending the small intestine of living dogs, rupture occurred on the convexity of the bowel, *i. e.*, the antimesenteric aspect, but that on distending

intestine removed from a dog ten hours after death, rupture occurred into the mesentery.

In the case of the living intestine, the coats yielded in the following order: (1) serous coat; (2) circular muscle fibers; (3) longitudinal muscle fibers; (4) mucous membrane.

Chlumsky adds that if tearing occurs in acute ileus, the peritoneal coat on the antimesenteric border of the bowel is the first part to yield.

In the light of clinical experience and Chlumsky's experimental results on the living animal, it can be stated without fear of contradiction that passive distension of the wall of the jejunum from pressure of its contents is not a factor in the causation of diverticula.

The unknown quantity in the production of intrajejunal pressure is the

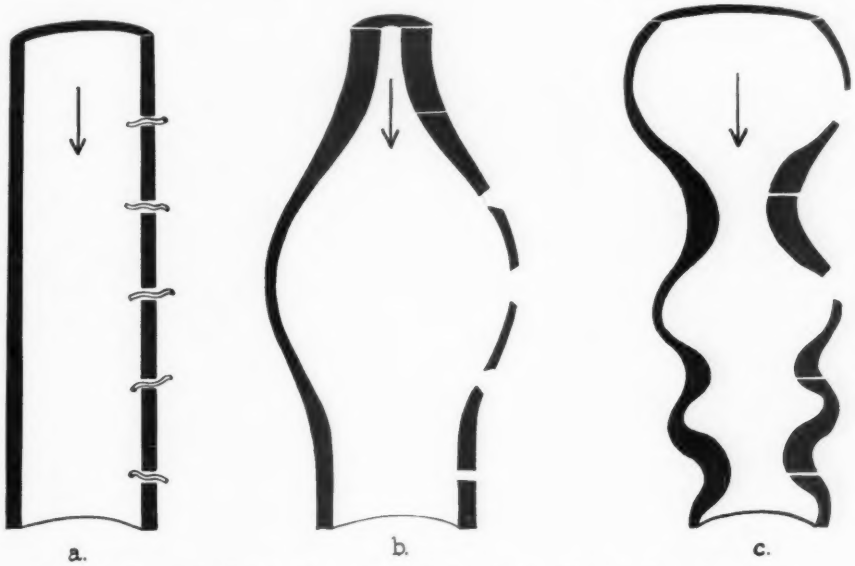

FIG. 15.—The mechanism of the formation of diverticula. Diagram illustrating normal contractions of the muscularis.

behavior of the muscular coat. Under normal conditions of contraction, as far as we understand them, a prolonged increased rise in pressure does not occur. One must postulate therefore some irregularity in muscular contraction. The better developed the muscular coat of the intestine, the more resistant the wall must be to passive pressure upon it. But if there is an irregular contraction of the muscular layers, so that one set of fibers is in spasm and the adjacent fibers are relaxed, a considerable alteration occurs from the normal both in the local pressure produced inside the lumen and the resistance of the wall to that pressure. Such local contractions and relaxations of the muscularis have been witnessed in the colon—anyone who stimulates the everted intestinal wall in a colostomy with prolapse will see them—and such a condition is therefore possible in the muscular coat of the small bowel.

Let us now examine the possible effects of regular and irregular contractions of the bowel's wall, as illustrated diagrammatically in Figs. 15 and 16. In Fig. 15 the muscular coat of the intestine is represented as a hollow tube, perforated at intervals for the passage of the blood vessels.

In Fig. 15 a peristaltic wave is suggested. The vascular gaps are narrowed in the contracting portion. During maximum contraction of the muscle, these gaps are temporarily obliterated, as they pass through two sets of muscle fibers contracting at right angles to one another. The peristaltic wave is preceded by a wave of relaxation. In this situation the vascular channels gape to their maximum extent, and constitute a very weak area in the bowel wall. Under normal conditions, however, there is no tendency for the mucous membrane to herniate through these areas, as the contents of the bowel are hurried along, and there can, therefore, be none but the most temporary rise of pressure inside the lumen of the bowel.

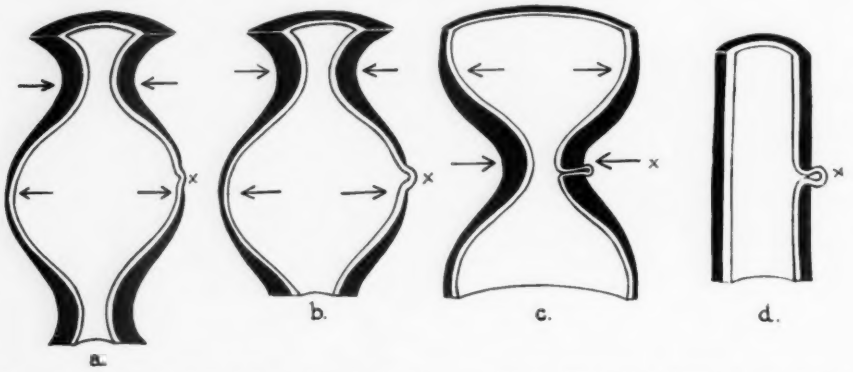

FIG. 16.—The mechanism of formation of diverticula. Diagram illustrating abnormal contraction of the muscularis.

If persistent irregular contraction of the bowel is present, then there may be considerable local increase in pressure. Such an irregular contraction is suggested in Fig. 16. Fixed spasm is occurring in two segments of the intestine, with the intervening segment relaxed (*a*). That spasm of the muscle to this extent can occur is shown roentgenologically in duodenal ulcer cases. As the contents are prevented from passing along the bowel, the pressure in the relaxed portion is increased, and the contents are being squeezed against the mucous coat, and tend to push between any interval that can be found between the muscle fibers. The vascular channels, gaping to their fullest in the relaxed muscle wall, offer the mucous membrane loci minoris resistentiae.

The probable stages in the formation of a diverticulum as a result of such persistent irregular contractions of the bowel are shown diagrammatically. A relaxed segment of the intestine lies between two contracted portions (*a* and *b*). As the result of the contraction, pressure is exerted upon the walls of the pockets. At (x) in the illustration, a vascular gap is suggested. As a result of the combination of increased pressure and

relaxation of the muscular wall, a wedge of mucous membrane is driven into the gap. If this part of the intestinal wall contracts, the wedge may be driven back, but there will come a time when the wedge driven out is of such size that the contraction of muscle about its base will consolidate its position by gripping it in the vascular gap (*c*). After many repetitions of this process, a permanent hernia of the mucous membrane will result (*d*). This will tend to increase in size purely as a result of the pressure inside the gut, for its wall is not supported by muscle, and will gradually be stretched by the constant pressure exerted upon it. It is difficult to say how long it takes for a diverticulum to develop to the size of those in the specimens described, but it is probably a matter of many years.

Diagnosis and Clinical Aspects.—The diagnosis of jejunal and ileal diverticula can be made only from roentgenologic examination, and then, usually, only if a residue of barium remains in the diverticulum after the jejunum has emptied. Contrary to the finding with regard to diverticula of the duodenum, jejunal diverticula are more frequently discovered at postmortem examination, when they are not likely to be missed unless of small size.

Symptomatology.—In only four cases was a diagnosis of diverticulosis made, and the clinical histories of these are as follows:

Case Reports

Case I.—A man, aged 49 years, was operated upon in 1929. In 1923, he experienced pain in the epigastrium two and one-half hours after meals. The pain was in the nature of a dull ache, and was to the left of the midline, but the patient could not localize it exactly. The pain was liable to come in attacks with free intervals. He occasionally vomited during an attack. Flatulence was a marked feature, and its occurrence coincided with the onset of pain. The appetite was good, but lately the patient had been afraid to eat. He had been constipated for the last three years.

Barium meal and roentgenologic examination revealed a large diverticulum of the jejunum just beyond the duodenojejunal flexure. It remained full for 24 hours after the jejunum was empty.

At operation the diverticulum was readily found six inches from the flexure. It opened from the mesenteric aspect of the gut, and hung downwards. The mouth was considerably narrower than the maximum diameter of the diverticulum, so that the latter was almost pedunculated. No other abnormality was found that could explain the symptoms.

The diverticulum was excised, the stump inverted, and the intestine closed by a continuous suture of catgut. On account of the narrowing of the lumen of the intestine caused by this procedure, a posterior gastro-enterostomy was performed. In May, 1931, the patient declared himself well, and had no further attack of the epigastric pain and flatulence.

A microscopic section of the wall of the diverticulum at the fundus shows a complete absence of muscle fibers. The base of the diverticulum was of no use from the standpoint of microscopy owing to the trauma caused by a crushing clamp.

Case II.—The patient is a man aged 69, first seen in November, 1930. Eighteen months ago he complained of pain in the lumbar region and slight pain of a vague character in the abdomen. At this time attacks of flatulence commenced, starting half an hour after meals and lasting from one to one and one-half hours. There was no nausea

or vomiting, and no loss of appetite or weight. He had been constipated for the last two to three months. The abdomen was distended without obvious cause. There was no tenderness but the left rectus appeared to be more spastic than the right. The benzidine test for occult blood was negative.

A barium meal and roentgenologic examination showed several moderate sized diverticula of the small intestine, best seen in the two hour film. Some of these remained filled four hours after the meal. There were also a few diverticula in the sigmoid.

The patient was not operated upon but was advised relative to regulation of bowels and diet. Seen six months later he appeared to be no better but still refused operation.

CASE III.—A man aged 67. For two years he had suffered vague epigastric pain, chiefly noticeable an hour or two after meals, and accompanied by marked flatulence. Belching appeared to relieve the pain. There was no nausea and no loss of appetite. He had been slightly constipated for many years.

Barium meal and roentgenologic examination showed the presence of two (? three) diverticula in the jejunum, best seen at two hours. There was no residue in them after the jejunum had emptied. Owing to age and general infirmity the patient was advised against operation.

CASE IV.—A woman, aged 38, had for six to eight years suffered from pain in the left hypochondrium. Flatulence occurred after meals to an extent which embarrassed the patient. She had suffered from constipation severely all her life. Roentgenologic examination showed the presence of several diverticula of the jejunum.

At operation, a segment of the upper jejunum was found to be somewhat dilated and the wall thickened as a result of hypertrophy of the muscular coat. Several diverticula were found on the mesenteric aspect. The length of bowel was resected (Fig. 5), and a side-to-side anastomosis made. Convalescence was satisfactory.

COMMENTARY.—The two outstanding symptoms common to these five cases are (1) vague abdominal pain, at an interval after meals, and (2) flatulence, corresponding in time incidence with the pain. That these symptoms can be definitely attributed to the presence of the diverticula is shown by the relief obtained from them after operation. They are probably due to retention of contents in the diverticulum and not to infection (diverticulitis). The one case in which acute diverticulitis was present will be described subsequently.

The literature provides a number of cases in which diverticulosis of the jejunum appears to have been responsible for symptoms. Tengwall[34] operated upon a woman of 51 who had many diverticula affecting the portion of the jejunum lying between a point 10 inches from the flexure and a point 30 inches below. They were diagnosed by roentgenologic examination before operation. The diverticula were situated on both sides of the mesenteric attachment, close to the mesentery but apart from it. The large ones showed depressions upon the surface as though composed of several diverticula of smaller size. He states that vague digestive disturbances with occasional nausea and irregularities of the bowel are usually met with in diverticulosis, but that the most common symptom is bleeding from the bowel. This latter statement is not borne out by the author's experience (but note the hematemesis in Braithwaite's[6] case). Gordinier and Sampson (1906), in describing a case of acute obstruction in a woman of 45, in whom 13 diverticula were present, state that they cannot find a single case in the

literature in which clinical symptoms arise from pressure due to jejunal diverticula.

Mackechnie's case in a woman of 43 with 13 diverticula of the jejunum, suffered from increasing constipation and flatulence, abdominal pain and attacks of vomiting. There were also three diverticula present in the duodenum. Lambert and Surmont[19] record a case of incomplete obstruction due to the presence of diverticula of the jejunum in a woman of 50. In Terry and Mugler's[35] case of a woman of 59, the early symptoms were due to a duodenal ulcer. In the 19 cases they review from the previous literature, only three gave symptoms.

Perhaps the most instructive case that has been recorded is that of Braithwaite.[6] A man of 54 had suffered from attacks of indigestion for a period of some nine years. The chief features were vague pain after meals, flatulence and borborygmus. Vomiting occurred frequently, and on one occasion was followed by five pints of unaltered blood. At operation four feet of the upper part of the jejunum were found studded with diverticula. A short circuiting anastomosis was performed without relief, and four months later the segment of bowel was resected, and relief from all symptoms was obtained.

It must be admitted that the symptoms of jejunal diverticulosis are not sufficiently characteristic to warrant a diagnosis of diverticulosis on clinical grounds alone, and roentgenologic examination is the final criterion. Caution must be exercised in holding the presence of diverticula as responsible for abdominal symptoms, and all other possible causes should be eliminated. If the author's own cases be taken in conjunction with those in the literature, it will be seen that the most common clinical symptoms are those of a flatulent dyspepsia. There may be in addition signs of incomplete obstruction, *viz.*, distension of the upper and left segment of the abdomen, with visible peristalsis. In Braithwaite's case, the upper part of the jejunum was distended and its wall thickened from hypertrophy of the muscle. Chronic diverticulitis giving rise to the peptic ulcer type of pain does not appear to occur in jejunal diverticulosis as in duodenal cases, for there is not a single case in the literature with a clinical history of this type. In the author's case of acute diverticulitis to be described immediately, the diverticulum was probably not of the pulsion type.

Acute Diverticulitis of the Small Intestine.—The specimen illustrated in Fig. 17 was removed at operation upon a male aged 53, who suffered acute abdominal pain. It consists of a segment of the small intestine. The bowel has been opened along the antimesenteric border, and a black glass rod passed through the opening of a diverticulum, which can be seen in contact with the mesentery. The diverticulum is spherical in shape and appears to be at some little distance from the bowel wall, communicating with the bowel lumen by a narrow opening. It is situated chiefly to one side of the mesentery. The peritoneal covering of the diverticulum and the adjacent portion of the intestine, and the mesentery, is a deep plum color from extreme congestion, and has lost its normal sheen. The wall of the diverticulum and of the bowel in the neighborhood is thickened

as a result of acute inflammatory edema. This is most marked in the diverticulum, and can be seen at the edges of the window cut for microscopic section.

The mucous membrane of the intestine near the orifice of the pouch shows the same inflammatory change. The mucous membrane of the diverticulum cannot be seen grossly, but its fate is shown in the microscopic section. Pus was present in the diverticulum when it was removed.

The wall of the diverticulum consists mainly of two coats, submucosa and serosa. The mucous membrane has been completely destroyed, leaving exposed a submucous coat which is infiltrated with inflammatory cells. The serous coat is enormously thickened by inflammatory edema, and is similarly, though less intensely, infiltrated. Between these two coats is a thin layer of muscle. The fibers are not clearly defined from the muscular coat, owing to the acute inflammation.

Clinical History.—A man of 53 had been suffering acute central abdominal pain for 24 hours. The temperature was 98° and the pulse 80. There had been no vomiting, and

FIG. 17.—Acute diverticulitis occurring in a solitary diverticulum of the small intestine (? ileum). Operative specimen.

no previous history of indigestion. The bowels had been opened prior to the attack. The recti were tense, and tenderness was most marked immediately below the umbilicus.

Laparotomy revealed a normal appendix, and a mass the size of a man's fist towards the center of the abdomen. This consisted of adherent omentum and coils of small intestine. Separation of these structures showed an acutely inflamed diverticulum. Ten inches of the intestine containing the diverticulum were resected and an end-to-end anastomosis made. The patient made an excellent recovery.

The author is in some doubt as to the exact nature of this diverticulum. Unfortunately, during the emergency operation the exact site of the diverticulum was not noted, but it was at some distance from the ileocecal valve, and yet comparatively low down in the abdomen. Presumably, therefore, the portion of intestine resected is either from the lower end of the jejunum or the upper end of the ileum. This is an unusual situation for a diverticulum of the pulsion type. It is probably an example of a congenital diver-

ticulum of the cystic type, and the following arguments are put forward in favor of this view:

(1) Its situation in an unusual position for a pulsion diverticulum.

(2) It has a well defined, though thin, muscular coat at its fundus.

(3) The diverticulum is almost a complete cyst, communicating with the lumen of the intestine by a very narrow opening.

(4) It stands a little way away from the wall of the intestine.

Diverticulum of the Jejunum Associated with a Malignant Growth of the Jejunal Wall.—This unique case has no exact parallel in the literature. The nearest approach to it is the duodenal pouch accompanying a growth which Morrison and Feldman described. The specimen (Fig. 11) was obtained at postmortem upon a woman of 53 who died from malignant disease, and consists of a segment of the upper portion of the jejunum.

There is a solitary diverticulum lying in contact with the mesentery. The diverticulum and the portion of intestine with which it communicates have been freely opened. The pouch is flask shaped with a thin wall containing no muscularis at the fundus, and has a fairly wide mouth. Apart from its situation, entirely to one side of the mesentery, it resembles in appearance a typical jejunal pulsion diverticulum. The intestine above it is somewhat dilated.

The wall of the intestine surrounding the mouth of the diverticulum is thickened, and its cut surface has a dense white homogeneous appearance, replacing the demarcation between the layers of the bowel which normally can be seen on naked eye examination. The microscopic section shows the submucosa to be infiltrated with large cells of a carcinomatous type. There is no tendency towards any definite order in the arrangement of the cells. There is very little stroma or evidence of active karyokinesis.

Other similar deposits of growth were found in the retroperitoneal region, the mediastinum, the pericardium and the diaphragm. This was the only evidence, however, of growth in the intestinal wall. The site of origin of the primary growth could not be determined. It was originally diagnosed as an "endothelioma," but further examination leaves little doubt that the cells of the growth are epithelial in origin.

The Treatment of Jejunal Diverticulosis.—The foregoing account shows how rarely diverticula of the jejunum give rise to symptoms. Also, in those cases where symptoms do arise, how hopeless any attempt at medical treatment is likely to be. The only possible way in which relief of symptoms may be obtained is by operative measures. Although care must be exercised before attributing abdominal symptoms to the presence of diverticula of the jejunum demonstrated roentgenologically, operation upon them is a far simpler procedure than operation upon diverticula of the duodenum. The jejunum has a mesentery, and is therefore free, which fact, together with its great length, makes any plastic or resection operation a comparatively safe and easy procedure. Therefore there need not be the same hesitancy in advising the patient to submit to operation as is the case with diverticula of the duodenum.

Operative Procedure.—Here, in my opinion, the issue is much clearer than is the case with diverticula of the duodenum. The best procedure, whether the condition be multiple or confined to a single pouch, is resection of the affected portion of the gut and end-to-end or side-to-side anastomosis.

NOTE.—My thanks are due to the Council of the Royal College of Surgeons for permission to publish this article, and to Miss Mary Barclay Smith who is responsible for the illustrations.

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THE PROBLEM OF THE LOW SIGMOIDAL GROWTH

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WITHIN the past decade the surgical treatment of neoplasms situated in the mobile portion of the sigmoid and in the rectum has reached a degree of standardization comparable to present day surgery for malignancy of the breast. The operations now in vogue vary somewhat in technical details and in scope, but the fundamental principle of all of them may be traced to three classic procedures, namely, the exteriorization operations of Paul[6] and Bloch[1] for sigmoidal lesions, the perineal excision of Allingham for low rectal and anal growths, and the radical combined abdominoperineal resection of Miles[4] for high rectal and rectosigmoid malignancies.

The changes made in these basic procedures have been directed for the most part toward a more extensive removal of regional lymphatic structures and toward increasing the scope of usefulness of the radical operation of Miles through the employment of multiple stage maneuvers in the poorer risk patients. For example, the obstructive resection (Rankin[7]), which is based on the early exteriorization measures, embodies the desirable features of the latter yet avoids the disadvantages, principally through immediate excision of the involved segment of bowel over a clamp devised for the purpose, thus permitting the removal of a greater expanse of mesentery without sacrifice of blood supply to the remaining parts. Moreover, the mortality both immediate, and remote, from metastasis, has been materially reduced by this procedure. In like manner, due chiefly to the efforts of Mummery,[5] the perineal excision has been changed from a purely palliative procedure to one of distinct usefulness in the cure of low rectal growths. He demonstrated that the peritoneum may be opened posteriorly and a considerable portion of the sigmoid with its vascular and lymphatic structures be excised without augmentation of the mortality. The combined operation of Miles, after more than a score of years, remains the undisputed ideal in the radical extirpation of cancers of the rectum and rectosigmoid. In the better risks and in the hands of surgeons highly skilled in such procedures, the one stage combined operation is still indicated but if all patients afflicted with carcinoma of the rectum and rectosigmoid were so treated the mortality would become prohibitive. Alternative measures based upon the surgical principles of Miles' operation have been proposed by various men with the modification that the operation be done in two or more stages. Jones,[2] of Boston, for many years has done a radical graded operation after a loop colostomy. One of us (Rankin[8])

modified Miles' operation in 1928 to a two stage procedure, and the following year Lahey[3] modified this operation still further by bringing the upper end of the rectal segment out of the lower end of the abdominal wound to be utilized for irrigation.

Neoplasms situated low in the sigmoid constitute a separate problem. They cannot be exteriorized without extensive mobilization of the rectosigmoid hence they are, as a rule, subjected to an abdominoperineal operation of some type. From the standpoint of curing the disease, such a course is entirely satisfactory but unquestionably there are instances in which a more conservative procedure, without sacrifice of the rectum, is justifiable. Our own indications for carrying out an anterior resection, of the obstructive type, with subsequent restoration of the bowel continuity are as follows: (1) small growths uncomplicated by infection; (2) growths of a low grade of malignancy, as determined by biopsy through the sigmoidoscope; (3) polypoid growths of doubtful benignancy; and (4) refusal of permanent colostomy. Unfortunately there are, not infrequently, factors present which make such a procedure inadvisable. Chief among these is a marked inflammatory reaction or abscess formation in the vicinity of the lesion, and instances of a short sigmoid flexure.

About a decade ago we became interested in anterior resection for selected cases of cancer of the rectosigmoid, but at first no attempt was made to reunite the divided ends of bowel. In this procedure a colostomy was established high in the sigmoid and at a subsequent operation the sigmoid, distal to the colostomy stoma and the rectosigmoid, were resected, the divided end of rectum inverted, and a new pelvic floor established. Our object at that time was to avoid the rather high mortality incident to the abdominoperineal operation and the morbidity which accompanies excision of the rectum. We felt then as we do now that the lymphatic structures draining the segment of bowel under consideration could be removed in a sufficiently radical manner by such a procedure in instances of early, low grade malignancy. In 1928, 26 consecutive cases were reported with but one death. However, in three instances failure in the blood supply to the inverted and retroperitonealized upper portion of the rectum necessitated removal of the rectal stump several days after the anterior resection. It was because of this uncertainty in the blood supply that we determined for growths situated in the rectosigmoid, below the peritoneal fold, that a combined maneuver would be more appropriate. For the low sigmoid growths we continued to employ a modification of the anterior operation in those cases in which there existed a long segment of sigmoid distal to the inguinal colostomy. Several weeks after establishment of the latter the involved portion of bowel was resected and the divided ends rejoined. At a little later period, in some of the cases, we applied the principle of exteriorization and in more recent years have utilized this method with increasing frequency and favor. This procedure, to which the term obstructive resection was applied by Rankin[9] in 1925, originally was employed for lesions affecting freely mobile segments of the colon, such as the transverse

FIG. 1.—Involved segment of sigmoid elevated several inches above its original position following division of peritoneal attachments of bowel and blunt dissection of rectum from hollow of sacrum. Point of ligation of sigmoid artery indicated. (Insert.) Rankin clamp applied to two limbs of sigmoid; the exteriorized portion to be removed with a cautery.

FIG. 2.—The peritoneum is brought under the loop and is sutured snugly around on all sides; sutures are not placed in the wall of the bowel.

colon and the midportion of the sigmoid. In the beginning two hemostats, or Payr clamps, were used to obstruct the bowel, but later a special three bladed clamp was devised with which to consummate the operation. This refinement in the technic was reported in 1929.

Our procedure varies little from that recently reported by David in which he applied the principle of obstructive resection to growths at the rectosigmoid.

Technic.—An incision is made low in the left rectus, or in the midline, after which the table is adjusted to the Trendelenburg position. Having packed off the small bowel in the upper abdomen, the sigmoid is isolated, and an incision made in the peritoneum of its mesentery on either side, from the base of the inferior mesenteric vessels forward to the bladder (Fig. 1). The sigmoid arteries, near their point of origin, are now ligated, care being taken at this time to identify the ureters, particularly the left. At this stage we have on several occasions ligated the superior hemorrhoidal artery without interference with the circulation to the divided ends of the sigmoid. We, believe, however, that this should not be practiced routinely since the collateral circulation through the middle hemorrhoidal arteries is not always adequate. Elevating the bowel, the hand is carried behind the intact superior hemorrhoidal artery, downward and forward into the hollow of the sacrum as far as the tip of the coccyx and in such manner by blunt dissection free all the tissue, including fat and lymphatic vessels in this region. The lateral incisions are now extended forward and the peritoneal attachments holding the bowel to the bladder are divided with scissors; here further blunt dissection completely frees the bowel anteriorly. There is seldom any bleeding of consequence following this dissection, but a hot pack placed in the pelvis will stop any venous oozing. It is now possible in most instances to lift the segment of bowel containing the growth outside the abdomen. This elevation of the growth, some 7 to 10 centimeters above its original position, is made possible by a combination of two factors, namely, division of the attachments at the rectosigmoid, and change in the shape of the rectum from a somewhat fusiform structure to a tubular one by virtue of the pull exerted on it in exteriorizing the growth (Fig. 1). The tendency of this diminution in the diameter of the rectum is to cause a desire for frequent defecation but in a relatively short period of time this sensation is overcome.

The next step is to excise a sufficient expanse of mesentery to insure removal of all lymphatic structures which might possibly drain the involved area. The special three bladed clamp (Rankin[11]) is then applied to the limbs of bowel proximal and distal to the growth, care being taken to insure good blood supply to the two ends of the bowel. In applying the clamp the handle should be toward the median line in order that subsequently the clamp may rest on the abdomen. Just anterior to this clamp another is placed, including both arms of the loop of bowel, and between these clamps the bowel is divided with a cautery (Fig. 1, insert). The rent in the mesentery is closed without putting sutures into the bowel. A new pelvic floor is constructed at a somewhat higher plane, about on a level with the promontory of the sacrum, in

order to peritonealize the elevated portion of rectum which is devoid of peritoneum. This is accomplished by employment of the lateral peritoneal flaps and if the patients are women, by utilization of the uterus and adnexa; although this maneuver is occasionally difficult, we have never found it impossible to accomplish satisfactory peritonealization. The clamp is now brought

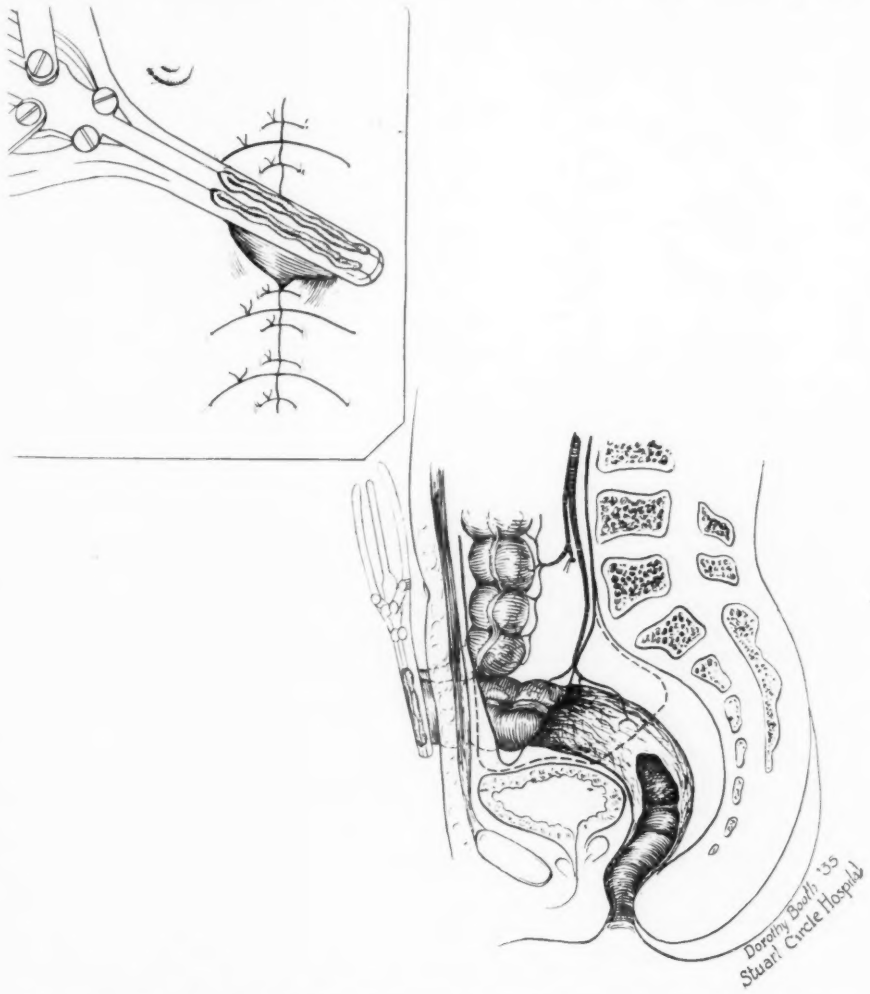

FIG. 3.—Sagittal section of the obstructive resection showing approximation of two limbs of bowel after removal of growth. The original pelvic floor, shown by dotted line, has been reestablished on a level with the promontory of sacrum in order to retroperitonealize the elevated deperitonealized rectum. (Insert.) Anterior view showing the clamp *in situ*, the proximal blade of which is opened in 48 to 72 hours in order to establish the temporary colostomy.

out of the wound at a point which leaves the structures loose. The wound is approximated snugly, close to the clamp, and a tongue of peritoneum is pulled between the two limbs of bowel, under the clamp; there is no need for sutures being placed into the bowel (Figs. 2 and 3). At times when the loop of sigmoid is very short and cannot be brought out of the wound readily, we do

not hesitate to leave the end of the clamp, grasping the bowel ends, deep in the wound. In such cases, however, iodoform gauze is wrapped around the bowel, beneath the clamp so as to avoid contamination in case of leakage. Noting that when the bowel ends were thus left deeply placed, beneath the muscles, spontaneous closure occurred in almost every instance following destruction of the colostomy spur, we have adopted this technic even in instances of growths situated in readily mobilizable portions of the bowel. At the selected time, the proximal blade of the clamp is opened, but the distal blade is left closed and the clamp is not removed; the clamp is permitted to drop off and this usually occurs about the seventh day after operation. Rather than tease open the agglutinated proximal end of the bowel on release of the blade it is better to allow the distention from pressure of gas within the colon to blow it open. The remainder of the operation is accomplished in a manner similar to that employed in the old exteriorization procedures; that is, the spur is cut out gradually with an enterotone, or with ordinary curved forceps, which are applied usually not earlier than two weeks or until the edema and inflammation commonly noted in the vicinity of the spur have disappeared (Fig. 4). After the lapse of a month or so the fistula is closed surgically, if necessary. In consequence of blunt dissection carried out in the hollow of the sacrum it has seemed advisable in most instances to institute drainage beside the rectum by introducing rubber tubing through a stab wound made just beneath the coccyx.

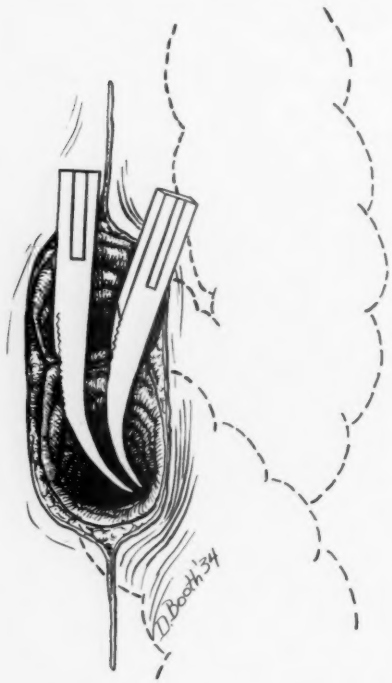

FIG. 4.—After the clamp has been removed and the wound is healed the spur is cut out of the two "gunbarrels" by application of clamps which effect a slow necrosis.

When it seems advisable, a cecostomy is established ten days to two weeks before the obstructive resection is performed. This necessity, however, has not often presented itself, for less than 10 per cent of the cases, numbering over 100, have been acutely obstructed, while in well over 90 per cent of cases it has been possible to adequately decompress the chronically obstructed bowel by a regimen that consists of initial purgation, repeated colonic irrigations, and an essentially non-residue diet. Strict avoidance of oral administration of barium in conjunction with roentgenoscopic examination of the intestinal tract when an obstructive lesion of the colon is suspected, has, we believe, reduced materially the necessity of preliminary cecostomy in our cases, for unquestionably such a practice is responsible for many instances of acute obstruction, superimposed upon a

chronic obstruction of long standing. Distention in these cases, even when the bowel has been left totally obstructed for 72 hours, has been of rare occurrence.

We fully recognize the tremendous value of preliminary cecostomy as an adjunct to surgery of the left half of the colon and rectum and believe that its employment is invariably demanded in the following instances: (1) acute obstruction; (2) chronic obstruction unrelieved by medical measures; and (3) resection of the left half of the colon followed by immediate anastomosis. Occasionally we also have availed ourselves of this measure in conjunction with the one stage combined abdominoperineal operations, even in the absence of obstruction.

The advantages of cecostomy as a measure preliminary to interval operations on the large intestines are manifold. Whipple,[12] in advocating its more frequent employment in such instances, has lucidly described these advantages as follows:

(1) It makes possible a proper cleansing of the colon before the major procedure.
(2) It permits the part anastomosed to be at rest until the period of fibroplasia is complete.
(3) It increases the comfort of the patient by reducing distention and pains of ineffectual peristalsis.
(4) It obviates the temptation and necessity in some cases of giving enemas or irrigations in the critical period of repair.
(5) In cases of partial or complete obstruction of the colon or rectum it has long been recognized as essential. If it works well in the patients who are desperately sick, it is even more efficacious in the non-obstructed case.

The necessity for preliminary cecostomy is diminished in a procedure such as obstructive resection, because the bowel continuity is not immediately restored after excision of the segment of colon that harbors the growth, the two ends of which are safely held in position by the two bladed clamp during the "critical period of healing" that takes place between the peritoneum and the bowel. On the other hand, it is usually indicated when the traditional Mikulicz exteriorization procedure is employed because decompression of the bowel by cecostomy permits edema of the extended limbs of colon to subside before the latter are excised.

A highly laudable proposition, that all cancers should be eradicated in the most radical manner possible is, unfortunately, not always practical. Many qualifying factors determine the feasibility of a given operative procedure. Among these may be listed age, sex, general condition of the patient, presence or absence of concurrent disease in other organs, relative degree of malignancy as determined by Broder's[10] index of malignancy, the age of the growth and extent of local lymphatic involvement and finally the skill and experience of the individual surgeon. Unquestionably it would be a mistake for every

patient with cancer of the rectum or rectosigmoid to be subjected to the operation of Miles or any other single procedure; and this holds equally true of the low sigmoidal growth. Nor should we base our selection entirely on the operative mortality and recurrence rate of a few surgeons whose experience with certain maneuvers is considerable. Given 100 cases of cancer of the rectum, one surgeon employing a less radical procedure may well have at the end of five years a larger number of patients living than another surgeon who has executed the most radical resection possible in each instance in a similar number of cases. Although we are convinced that the procedure here described may be advantageously employed in certain carefully selected cases, we do not lose sight of the fact that even in instances of a low sigmoidal growth the ideal procedure, in the absence of contra-indications, is the combined abdominoperineal operation.

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CARCINOMA OF LINITIS PLASTICA TYPE INVOLVING THE INTESTINE*

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THE term "linitis plastica" is now generally considered to designate a carcinomatous process in which the stomach is more or less diffusely contracted and has thickened and relatively inflexible walls. Available data, however, it seems, do not preclude the possible occurrence, rarely, of a benign inflammatory form of the disease. It appears that Liéutaud, in 1779, may have recognized the condition; however, credit for reporting the first case is usually given to Andral. Since Andral described the condition as a simple type of hypertrophy, in 1829, some 42 names have been ascribed to the lesion (Howard[13]). Brinton,[5] in 1865, called it linitis plastica (Greek meaning linen cloth or net; Latin, *ex lino factum*), because of the network arrangement of the connective tissue in the gastric submucosa. Despite the fact that Brinton considered the lesion benign, and despite the merit of the criticisms of the term, it has persisted to date as the most generally used name for the condition. For this, if for no other reason, we feel that we are probably justified in its use to facilitate discussion herewith. The term "leather-bottle stomach" (Stretton,[23] 1909), also frequently used, is hardly appropriate for extragastric manifestations of this affection.

Although linitis plastica confined to the stomach is known to occur not infrequently, relatively few cases of this process occurring in the intestine have been cited. Coe,[7] in 1931, reporting a case in which there was involvement of both flexures of the colon and of the rectum, stated that he had seen no other similar cases reported in textbooks or in the radiologic literature. We have, however, in a search of the general literature pertaining to the subject, encountered reports of 37 cases in which there was involvement of one or more portions of the intestinal tract. In only one of these, the primary coexistence of the gastric lesion apparently was not noted. This was a case reported by David,[9] in which the rectum was involved, and it was not stated that the stomach was examined. Our series includes six cases of linitis plastica involving the intestinal tract, obtained from the files of the Mayo Clinic.

CASE REPORTS

CASE I.—A man, aged 34 years, came to the clinic because of stomach trouble of 12 years' duration. He gave a history of epigastric distress, coming on two hours after

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meals, which at first had been relieved by taking soda. As time went on, the distress had become more constant and had been accompanied by vomiting. The vomiting had increased in frequency until the time of the patient's admission to the hospital. On examination, the abdomen was found to be distended. Analysis of gastric content revealed total acidity of 50 and free hydrochloric acid of 30 (Töpfer's method); the total quantity of gastric content recovered measured 450 cc. The concentration of hemoglobin was 73 per cent. The Wassermann reaction was negative and results of roentgenologic examination of the stomach appeared to be negative.

Despite the lack of positive roentgenologic evidence, the patient's persistent symptoms of pyloric obstruction warranted operation, it was felt, and exploration was performed. This revealed diffuse thickening of the pyloric end of the stomach, of the second portion of the duodenum, and of the first six inches (15 cm.) of the jejunum. Gastro-enterostomy was performed. The patient is living today, 11 years after operation. He writes that he still has some stomach trouble.

Case II.—A woman, aged 42 years, complained of a "filled-up" sensation in the stomach, of 16 months' duration. The distress usually occurred half an hour before meals and was relieved by vomiting. She said she felt most nearly well when eating small amounts of soft food. Her appetite was normal and her bowels moved regularly. She had lost 14 pounds (6.5 Kg.). Analysis of gastric contents revealed total acidity of 26, free hydrochloric acid of 8, and a total quantity of 50 cc. The Wassermann reaction of the blood and of the spinal fluid was negative. The concentration of hemoglobin was 64 per cent. The roentgenologic diagnosis was a lesion of the middle third of the stomach, possibly gastric syphilis. Other findings were irrelevant.

At exploration, an inoperable, diffuse carcinoma of linitis plastica type, involving the stomach and the central portion of the transverse colon, was found. The patient died one month and a half postoperatively, after leaving the hospital.

Case III.—A woman, aged 52 years, entered the clinic because of abdominal distress of seven weeks' duration. She complained of abdominal pain which was most marked in the right lower quadrant, vomiting with gradual inability to take solid foods, and diarrhea. The abdomen was distended and tender. The value for hemoglobin was 75 per cent, and erythrocytes numbered 6,790,000 per cubic millimeter. The high count was probably attributable to concentration of blood incident to dehydration. Analysis of gastric content and roentgenologic examination of the stomach were not made because of the obvious intestinal obstruction.

Palliative ileosigmoidostomy was performed for what appeared to be a diffuse carcinoma involving most of the colon. The patient died one week after operation. Postmortem examination revealed a carcinoma, Grade 4, of linitis plastica type, involving the entire stomach to within 2 cm. of the pylorus. The identical type of lesion produced a number of constrictions in the small intestine, causing obstruction, in addition to diffuse thickening of the entire colon. The esophagus, right oviduct, and peritoneum were also involved.

Case IV.—A man, aged 50 years, complained of nausea and vomiting occurring after meals. His appetite was normal, but he was afraid to eat because of the resultant distress. The discomfort was relieved by vomiting. He had lost 15 to 20 pounds (7 to 9 Kg.) and appeared considerably emaciated. An epigastric mass was palpable. Analysis of gastric contents revealed total acidity of 60, free hydrochloric acid of 40, and a total quantity of 2,080 cc. The concentration of hemoglobin was 71 per cent. The Wassermann reaction of the blood was negative. Roentgenologic examination revealed a carcinoma of the pyloric end of the stomach, with retention and enormous dilatation.

Exploration revealed diffuse thickening of the entire stomach, and pyloric obstruction. A palliative gastrectomy and posterior Pólya anastomosis were performed. The patient died two months after operation. Postmortem examination revealed a linitis plastica type of carcinoma involving what remained of the stomach. The colon, rectum,

jejunum and bladder were likewise involved. The contributory cause of death was a pulmonary embolism.

CASE V.—A man, aged 49 years, complained of persistent vomiting and loss of appetite of three months' duration. His strength had failed and he had lost 16 pounds (7 Kg.). An epigastric mass was palpable. Analysis of gastric content revealed total acidity of five, no free hydrochloric acid, and a total quantity of 275 cc. Roentgenologic examination of the stomach gave evidence of a carcinoma involving the pyloric region.

Exploration revealed an inoperable carcinoma of the stomach, head of the pancreas and common bile duct. The patient died two months after operation. Postmortem examination revealed a carcinoma of linitis plastica type involving the stomach, transverse colon, mesentery, peritoneum, appendix, lymph nodes, pancreas, and ureters.

CASE VI.—A woman, aged 50 years, entered the clinic because of intestinal obstruction. She gave a history of stomach trouble of two and one-half years' duration. Her appetite had been poor. She was nauseated after eating. Prior to the onset of her trouble,

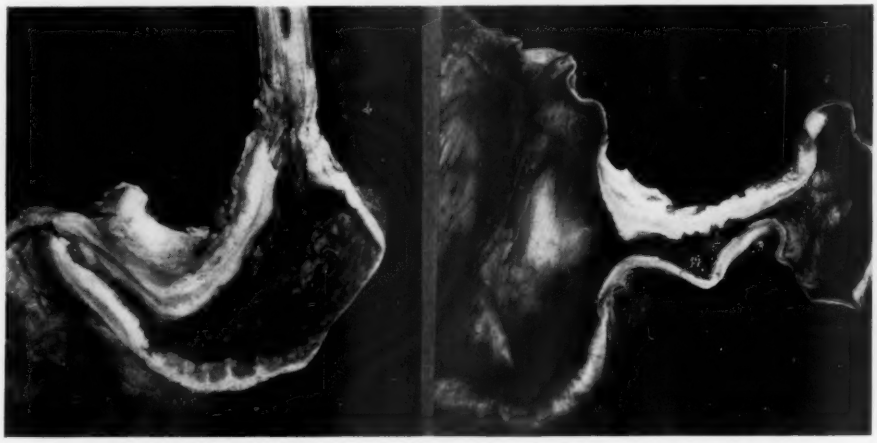

FIG. 1.—(Case VI.) Typical linitis plastica of stomach. A diffuse, fibrous type of thickening of the gastric wall is evident.

FIG. 2.—A type of lesion identical with that represented in Fig. 1, involving the splenic flexure of the colon. Huge dilatation of the ascending colon is present.

she had received roentgenologic treatments for a fibroid uterus. A roentgenogram, made elsewhere a year before, was said to give evidence of pyloric obstruction. For two weeks she vomited almost continually and had intermittent abdominal cramps. She had had no bowel movement for one week. She was emaciated, having lost some 35 pounds (16 Kg.) since the onset of her illness. Abdominal distention was marked. Active peristalsis of the small bowel was visible. Other findings were irrelevant. Roentgenograms were not taken.

A clinical diagnosis of intestinal obstruction of the lower part of the ileum or the colon was made. Transnasal duodenal suction was instituted and solution of sodium chloride and glucose was administered by vein for several days preparatory to operation. The patient's general condition improved and the blood chlorides were brought up to 611 mg. per 100 cc.; then exploration was made. Operation revealed two constrictions of the colon, at the hepatic and splenic flexures, appearing to be about two inches (5 cm.) in length. The ascending colon and ileum were markedly dilated. The stomach was thickened but the prominent gross pathologic change was of the colon. Palliative ileocolostomy was performed to relieve the obstruction. The patient died of peritonitis on the ninth postoperative day. Postmortem examination revealed a linitis plastica type of carcinoma involving the entire stomach, the hepatic and splenic flexures of the colon as described, the esophagus, and the ileum. Microscopic examination revealed involvement

of the ascending colon, bladder, perirenal fat and diaphragm; involvement of these structures was not grossly evident (Figs. 1, 2, 3 and 4).

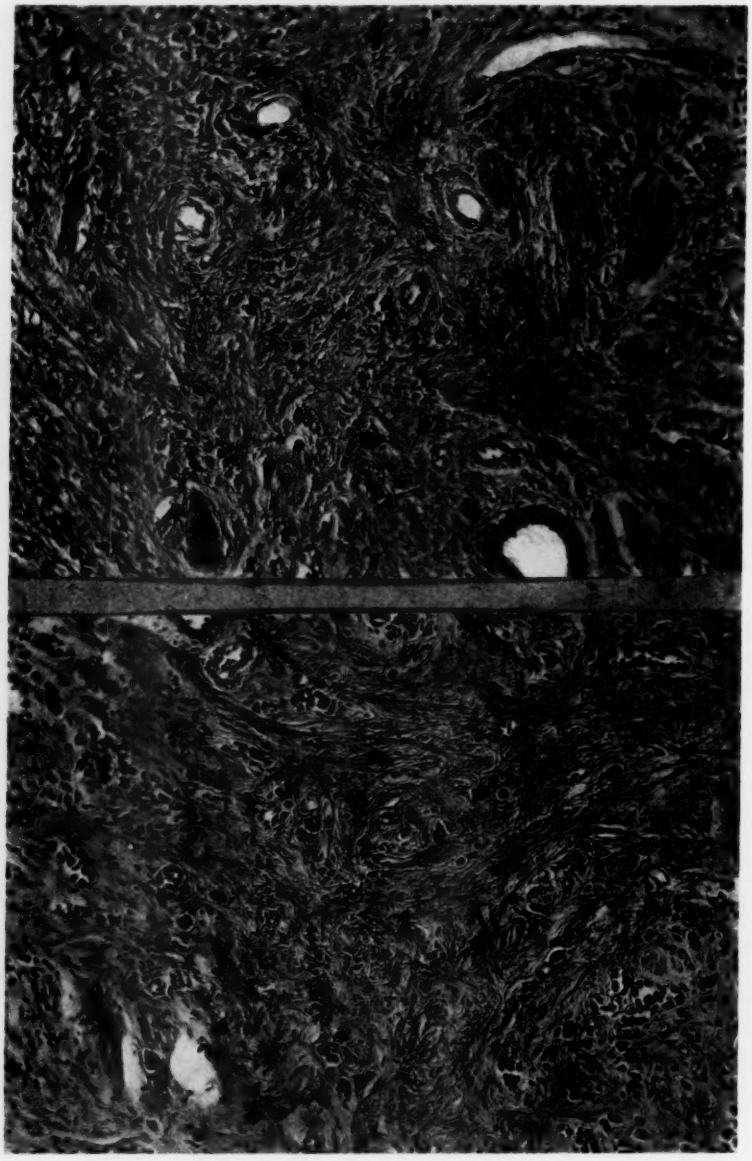

FIG. 3.—(Case VI.) Typical histopathologic section of gross specimen of stomach represented in Fig. 1. The so called network arrangement of connective tissue, with interspersed malignant cells, may be noted.

FIG. 4.—(Case VI.) Section taken from gross specimen of colon represented in Fig. 2. The pathologic change is obviously the same as that in the stomach represented in Fig. 3.

CRITIQUE OF CASES

Five cases of our group were encountered in reviewing 235 explorations in which linitis plastica of the stomach was diagnosed. This represents the total

number of such cases diagnosed in more than 11,000 exploratory operations on the stomach. Four cases came to necropsy and represent the number of times intestinal involvement occurred in 36 cases of carcinoma of the linitis plastica type encountered in some 4,000 routine postmortem examinations. The proportionately greater frequency of intestinal linitis plastica, postmortem, in our group of cases is probably accounted for by two factors: namely, the patients are seen later and therefore metastasis would be more likely to be present, and, examination of the viscera at operation is more difficult than at the necropsy table. This suggests that probably a relatively greater number of cases exist, in which there is intestinal involvement, than we are now aware of. In all of the four cases in which postmortem examination was performed, death occurred in the immediate postoperative period; three are included in the five surgical cases, but one is not, for the gastric lesion was not discovered at operation (Case III).

Unusual Manifestations.—The patient of Case III, as well as the one on whom we operated, came to operation because of symptoms of intestinal obstruction which were so prominent that what gastric symptoms were present, if any, were masked.

Some question may be raised as to the authenticity of Case I, as unfortunately we have no sections of the lesion. However, at operation the diagnosis of linitis plastica was made, and after carefully studying the case it seemed to present the characteristics of this condition. We are at a loss in attempting to place it in any other group. Aside from the unique situation of the lesion, this case is of especial interest in view of the fact that the patient is still living 11 years after operation. Other more bizarre situations in which what appeared to be linitis plastica was present have been recorded. Weber[25] reported a case in which the primary pathologic change seemed to be in the gallbladder, without evident involvement of the stomach, and Albot and Michaux,[1] a case in which acanthosis nigricans was present.

Ages.—The ages of the patients in this series ranged from 34 to 52 years. The average age was 46 years. Only one of the patients was less than 42 years of age. The ages of patients who had linitis plastica (not confined to those who had intestinal involvement) whose cases are reported in the literature have been somewhat higher than those of our patients. The average age of Lyons' patients was 52 years. Friedewald and Morrison's[11] ranged in age from 26 to 56 years; Baumgartner, Case and Deegan's[2] from 20 to 80 years, and Lyle's[16] from 40 to 60 years.

Sex.—Of our patients who had linitis plastica involving the intestine, three were men and three were women. The authors mentioned in the foregoing paragraph reported that linitis plastica occurred about twice as often among men as among women in their cases.

Situation.—In all of our cases the primary lesion was found in the stomach. In one case the duodenum was involved throughout its second part. The small intestine, exclusive of the duodenum, was involved in five cases and the colon in five cases. There was a typical lesion of the rectum in one case.

In four cases multiple metastatic growths, of linitis plastica type, were demonstrable, exclusive of the involvement of the large and small bowel. These lesions were present in the diaphragm, pancreas, perirenal fat, esophagus, appendix, peritoneum, omentum, mesentery, fallopian tube, uterus and bladder. The situation of the lesions in our series of cases corresponds roughly to that of those we have found reported in the literature.

Pathology.—Although divergent views have existed as to the nature of the pathologic change in linitis plastica, it is now generally conceded that it is a malignant condition. Wyard[26] went so far as to say that it is a gross entity only and that a number of conditions may cause leather-bottle stomach. Brinton[5] wrote that the lesion was inflammatory, although it seems he was not sure of this. Krompecher[15] and Curtis[8] expressed the belief that it is a form of gastro-intestinal sclerostenosis, and Pauchet,[20] Bensuade, Rivet and Lewald,[4] Holmes and Ruggles,[12] and others, gave as their belief that some of the lesions are syphilitic. The weight of evidence now indicates that linitis plastica is a small cell carcinoma (MacCarty[18]). Broders[6] likewise feels that so called linitis plastica is an adenocarcinoma and that it is merely one of the scirrhous type which has a peculiar tendency to form an excessive amount of fibrous tissue. Ewing[10] concluded that it is a peculiar form of gastric carcinoma with cells of limited capacity for growth, occurring in a resistant subject. These factors, he maintained, tended to spontaneous regression of the tumor process. He expressed the belief that the rare cases in which the patients recover after gastro-enterostomy represent such spontaneous recovery. The patient who survived after exploration in our group (Case I) may fall into such a category, although we cannot prove that in this case the growth was not benign.

The pathology of linitis plastica involving the intestine is, obviously, essentially the same as that of the stomach. Grossly the bowel, as the other involved organs, appears waxy, lusterless, and pale because of disturbance of the blood supply attributable to perivascular fibrosis. The intestinal wall is diffusely thickened and relatively inflexible, which produces varying degrees of contraction of the lumen. Sectioning the bowel gives the sensation of cutting gristle. The mucosa may be destroyed or may appear intact. Ulceration did not occur in any of our cases, although it is occasionally present in the gastric lesions (Lyons[17]). The extent of involvement ranged from narrow, discrete, annular constrictions of the bowel to involvement of numerous portions of the small intestine and thickening of the entire colon.

Microscopically the fibrous tissue characteristically arranges itself in more or less of a network and this, as has been mentioned by others, is most marked in the submucosa, although all layers of the stomach or intestinal wall may be involved. Scattered among the areas of fibrosis are cancer cells with large nuclei which are often vesicular in appearance. At times signet ring forms are evident. The number of mitotic figures varies according to the degree of malignancy. In the experience of Broders, most carcinomas of this type are of high grade of malignancy. In Case III of our series the lesion was Graded 4, according to Broders' index of malignancy. The cancer cells may be numer-

ous and may be in definite glandular arrangement, or they may be so obliterated by fibrosis that only careful and repeated study of numerous sections will reveal them. It is this factor largely which prompted Ewing, Lyons, Howard, Miller,[19] Beath[3] and others to feel that most, if not all, of the growths in so called benign cases reported were in fact cancer. We concur in this opinion. Such cases in which the growths were originally diagnosed as benign, and which were subsequently rechecked carefully and were found to be malignant, have been reported by Telger,[24] Streicher,[22] Singer,[21] David, Lyons, and others. All of the sections studied in our group were of malignant tissue.

Symptoms.—The symptoms are primarily those of linitis plastica of the stomach, which are similar to those of any slowly growing gastric cancer. Epigastric distress, which usually is worse after meals, sometimes relieved by taking soda and usually by vomiting, is the cardinal symptom. As the disease progresses and the size of the stomach diminishes, smaller and smaller amounts of food can be eaten. There is usually loss of weight and strength, and anorexia after the condition has advanced. When intestinal involvement becomes marked, the essential symptoms referable to the bowel are those of obstruction, and these may obscure the gastric pathologic condition. This occurred in two cases of our group. Duration of symptoms in this series ranged from seven weeks to 12 years.

General physical examination may or may not be helpful. Abdominal distention was evident in three of our cases and an epigastric mass was palpable in two instances. One of these was unusual in that instead of the stomach being contracted, it was hugely dilated because of pyloric obstruction. Achlorhydria was present in but one case, free hydrochloric acid of eight occurred in one case, and analysis of gastric content in the other two cases disclosed normal acidity. Anemia was only moderate, the readings of hemoglobin varying from 64 to 89 per cent. Dehydration and concentration of blood may have elevated these values.

Diagnosis.—It is difficult if not impossible to diagnose linitis plastica clinically with a reasonable degree of certainty (Streicher[22]). The difficulty in recognizing the primary gastric lesion as a linitis plastica type of growth has been stressed frequently. Lyle cited only two correct diagnoses in 126 cases, and Lyons six in 38 cases. Kirklin[14] expressed the opinion of most roentgenologists, namely, that one cannot hope to do more than diagnose the gastric lesions as carcinoma; to attempt to specify whether or not they are of linitis plastica type is impossible. Coe stated that the most outstanding roentgenographic feature is the rapid emptying time of the stomach. He described the usual outline of the stomach as that of a narrow inverted cone. Others have described it as being sausage shaped (Lyle, Baumgartner, Case, Deegan and Coe). Coe reported a case in which the gastric and colonic lesions were diagnosed preoperatively. We know of no other instance in which linitis plastica involving the colon has been diagnosed before operation. This applies to our six cases. In two of them, however, the diagnosis of low intestinal obstruction was obvious preoperatively.

Probably the most that can be said concerning the diagnosis of the intestinal lesion is: given a patient presenting a history and findings suggestive of linitis plastica of the stomach and who develops intestinal obstruction, consider the possible existence of an intestinal lesion of this type.

Prognosis.—Linitis plastica which has metastasized to the extent of involving the intestine is fatal. Rare cases, such as Case I of our series, are excepted and it must be kept in mind that sections for microscopic study were

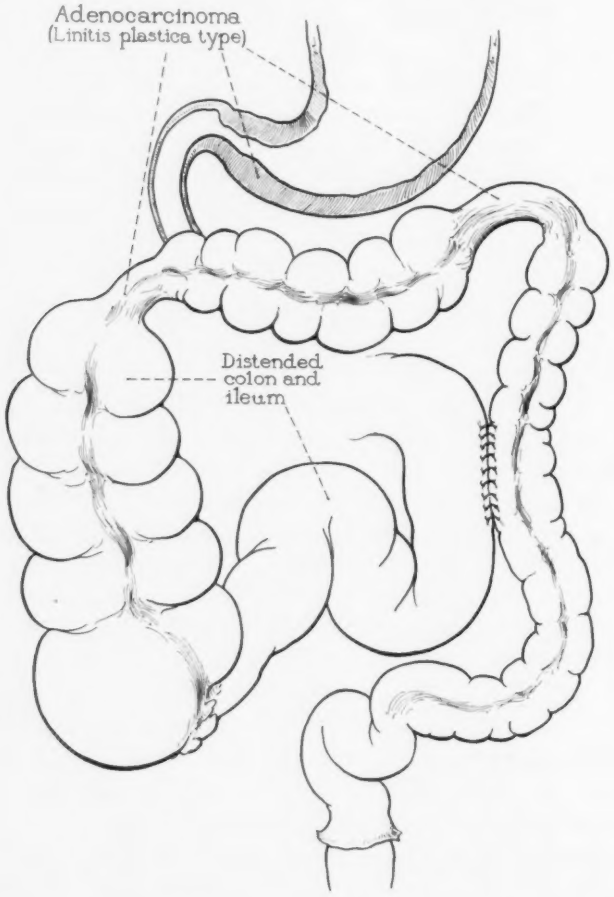

FIG. 5.—Ileocolostomy for palliative relief of obstruction, the degree of which is shown by dilatation of the ileum and ascending colon.

not available in this case. If the growth in this case was not benign, it must be a manifestation of spontaneous cure of cancer of which Ewing has spoken.

From the onset of symptoms (except in this one case) our group of patients lived from eight weeks (case with lesion of Grade 4) to two and one-half years.

Treatment.—Other than palliative treatment of linitis plastica of the intestine is futile since, as is evident from a review of our cases and of those

reported in the literature, multiple metastatic growths usually are present by the time the condition has progressed to a stage at which the pathologic change involves the intestine. Patients who are not immediately moribund, who are in fairly good general condition, and whose intestines are obstructed, probably deserve the palliative procedures of entero-anastomosis, ileosigmoidostomy, enterostomy, colostomy, or ileocolostomy (Fig. 5). Such types of operations were performed in our cases.

SUMMARY AND CONCLUSIONS

An effort has been made to review in some detail six cases of carcinoma of linitis plastica type involving the intestine. These cases, with the 37 such cases of which we have found reports in the literature, bring the total number to 43 reported to date.

Available data suggest that, although no doubt rare, the condition probably occurs with greater frequency than the number of cases reported would indicate. Although our group of cases as a whole is of interest chiefly because of the rarity of the lesion, two cases are of more interest because of the prominence of symptoms referable to the colon, namely, obstruction, and one case is of especial interest because the patient is still alive eleven years following exploration. The growth in this case may be benign, although the question of spontaneous cure of cancer arises. Diagnosis clinically is difficult. In all of our sections studied microscopically, malignancy was demonstrated. Without exception the primary lesion was found to be in the stomach.

In view of the usual existence of multiple metastatic growths, in addition to the presence of the gastric lesion when linitis plastica has reached the stage of intestinal involvement, other than palliative forms of treatment are futile.

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ACUTE REGIONAL ILEITIS

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CROHN, GINSBURG, AND OPPENHEIMER'S[1] description of regional ileitis (1932) has been followed by a number of confirmatory publications in the American and foreign literature.[2, 3, 4, 5, 10, 11, 12, 13, 15, 16] This disease may be described as a chronic, hyperplastic, nonspecific inflammation of the lowermost section of the ileum, extending to and stopping at the ileocecal valve, and usually accompanied by an involvement of the adjacent mesenteric lymph nodes. Frequently more acute reactions supervene, which may lead to ulceration, even fistula formation, in the bowel wall and to suppuration in the lymph nodes. The pathologic changes appear, as a rule, severe and irreparable, so that the excision of the diseased intestines has been advised by some as the rational therapy.

With the increased recent attention given to this condition, observations have been accumulating which show that both the pathologic picture and the clinical course of these inflammatory processes may show considerable variations. Concomitantly, the surgical treatment has been varied, and the results obtained in several cases seem to prove that such a formidable procedure as a bowel resection is not always necessary.

It is this variability of the inflammatory process and the uncertainty concerning the indications for surgical procedures which prompt us to report three cases which have come under our observation. While most of the previous reports on regional ileitis emphasize the gradual onset and the chronicity of the symptoms, the clinical manifestations in our experience were those of an acute surgical abdomen urgently demanding exploration. The appearance of the inflamed bowel in these cases conveyed the impression that the inflammatory process was acute and probably transitory, not precluding a restitution of the diseased tissue. Accordingly, a minimal amount of surgical interference was attempted: in two cases only recourse was taken to a temporary deviation of the intestinal contents by an enterostomy. The result of this treatment was satisfactory. In one of these cases we could convince ourselves by a microscopic examination of the previously diseased ileum removed at a subsequent operation that a complete anatomic restoration had taken place. In the second case a similar spontaneous repair may be surmised in view of the patient's continuous freedom from symptoms. The third case is still too recent to permit an opinion concerning the final outcome.

CASE REPORTS

CASE I.—(No. 250506.) A boy, five and one-half years old, entered the Jewish Hospital complaining of abdominal cramps of six hours' duration. The pain came at intervals

and was, almost from the beginning, accompanied by vomiting. An hour after onset of the pain an enema had resulted in the evacuation of a formed stool.

In this clinical history the following details are noteworthy: Three years ago he supposedly had a similar though milder attack of abdominal pain. About the same time he had suffered from a persistent cervical adenitis with periods of recurrent fever, on account of which the adenoids were removed and, after this had no effect, two enlarged lymph nodes were excised.

The temperature on admission was 99° (rectal); pulse, 80; white blood cells, 19,000, with 92 per cent polymorphonuclears and 8 per cent lymphocytes. On physical examination an abdominal mass was seen and felt 2 cm. to the right of the umbilicus. Peristaltic waves could be observed traversing the abdomen and apparently stopping at the mass. On rectal examination no obstruction or tumor could be felt, but a definite amount of blood was expelled on withdrawal of the finger. Urine normal.

In view of these findings the diagnosis of intestinal intussusception, probably ileocecal, was made and an immediate laparotomy performed. The peritoneal fluid was not noticeably increased. The last 15 cm. of the ileum was much swollen, purplish red, and the serosa appeared finely granular and spotted with numerous petechiae. The mesentery of the involved section of the ileum was very edematous and contained many large lymph nodes, some attaining a diameter of 2.5 cm. No thrombosis was found in the mesenteric vessels. The cecum and appendix were apparently normal. The pathologic process stopped abruptly at the ileocecal junction. There was no invagination of the ileum into the cecum nor ecchymotic or abrasive marks suggesting a recently released intussusception. We considered the inflamed bowel segment viable and believed that a temporary deviation of intestinal contents would influence the inflammatory process favorably. A rubber tube was inserted into the ileum after the method of Senn, about 10 cm. above the ileocecal valve, still within the proximal limit of the involved intestine. The appendix was removed and one of the largest lymph nodes excised for microscopic study. The abdomen was closed and the enterostomy brought up close to the incision and fixed to the peritoneum.

The microscopic examination of the appendix showed no inflammatory changes. In the lymph node a marked inflammatory hyperplasia was found, but no indication of any specific infection.

The postoperative course was complicated. Fever persisting until the twenty-fifth postoperative day. On the second day after operation the patient passed a large amount of dark blood by rectum. On the fifth postoperative day the incision broke down, after which a considerable herniation with eversion of the enterostomy developed. Most of the intestinal content nevertheless found its way into the colon.

Three months after the operation the patient seemed in a satisfactory condition for the closure of the intestinal fistula. The appearance of the terminal ileum did not show any traces of the previous extensive inflammatory swelling. The lymph nodes in the ileocecal region, although still enlarged, were less than half the size of three months before. A short section of ileum containing the fistulous opening was excised and the continuity restored by an end-to-end anastomosis.

The microscopic examination of the resected piece of ileum revealed only blood extravasations and a minimal round cell infiltration in the subserous layer, which could easily be accounted for by the exposure of the bowel wall in the infected wound.

CASE II.—(No. 321945.) A male student, 27 years of age, entered the Jewish Hospital with the provisional diagnosis of acute appendicitis. After having partaken of food and drinks excessively the previous night, he was awakened by a moderately severe pain in the epigastrium which later localized in the lower part of the abdomen, slightly more on the left side. Prior to admission he had vomited four times. His bowels had moved after an enema. The patient stated that he had a similar attack of pain accompanied by nausea and vomiting only five days previously which kept him in bed for two days. His physician suspected at that time an acute appendicitis.

Examination of the abdomen revealed distention in the lower half. There was no visible peristalsis. Tenderness was quite diffuse, but more in the left lower quadrant. Distinct rebound pain was present on both sides. The rectal temperature was 102°; pulse, 110; white blood cells, 23,000; respiration, 22. The differential count showed 21 per cent stabs, 71 per cent polymorphonuclears, 3 per cent lymphocytes and 5 per cent monocytes.

Under the assumption that the patient was suffering from an acute appendicitis or diverticulitis, a laparotomy was immediately performed. The peritoneal fluid was found markedly increased and cloudy. The last 15 cm. of the terminal ileum was swollen, dark red and edematous. Its serosa was roughened and covered with shreds of fibrinous exudate. While the proximal limit of the inflammatory swelling was somewhat indistinct, the process stopped abruptly at the ileocecal junction. The mesentery adjacent to the involved part of the ileum was infiltrated, but did not contain any thrombosed vessels or blood extravasations, nor were its lymph nodes enlarged. The intestinal contents could be made to pass from the slightly dilated small intestines above the lesion through the involved segment into the cecum by gentle pressure and a milking manipulation. Nothing indicated a volvulus or strangulation. An enterostomy of the ileum just proximal to the lesion was made by inserting a catheter according to the Witzel technic. Three Penrose tubes were placed around the inflamed ileal loop because of the presence of a peritoneal exudate.

After the sixth postoperative day the temperature dropped from a peak of 104° and the patient recovered quickly. The intestinal fistula closed spontaneously a few days after removal of the tube.

The patient was reexamined two years after the operation. He complained of an occasional mild pain in the abdomen, not well localized, which was never severe enough to interfere with his daily routine or to make him consult a physician.

Case III.*—The patient, a housewife 24 years of age, complained of mild abdominal cramps and occasional nausea for about five days. She had not interrupted her daily routine and attributed her discomfort to impending menstruation. On the day of her admission to the hospital the menstruation had ended, but her pain slightly increased. In the afternoon the temperature rose to 102°. Vaginal examination excluded an adnexal inflammation. The abdomen was soft, but tender in the right lower quadrant. In this area was also a rebound pain. The W. B. C. was 14,000; the differential count, 6 per cent stabs, 70 per cent polymorphonuclears and 24 per cent lymphocytes. Urine negative. The impression of all consultants was that these findings indicated an acute appendicitis, retrocecally situated.

At operation the appendix was found thickened, edematous, but not acutely inflamed. The last six inches of the ileum were red, markedly swollen and stiffened. The mesentery was not thickened and did not contain palpable lymph nodes. The appendix was removed, the uterus and adnexa explored and found normal. The patency of the ileocecal valve was assured by careful palpation between two fingers. The abdomen was closed without drainage.

Microscopic examination of the appendix: The mucosa is everywhere intact. The lymphoid tissue is rather prominent. The subserosa is edematous. Bacteriologic culture from cross section of appendix: *Bacteria proteus valerii* in pure culture.

The postoperative course was short and uneventful. For about five days blood could be detected chemically in the feces. The patient left the hospital apparently cured on the ninth postoperative day. While still continuing on a diet free of roughage, patient has at present no complaints.

Discussion.—Acute inflammatory processes in the terminal ileum were mentioned by Crohn[1] and co-authors in their original description. They con-

*We are obliged to Doctors Myer, Sale and Ehrenfest for permission to report this case.

sidered these conditions to be acute episodes or abortive stages of the hyperplastic and chronic regional ileitis. Clute,[2] Ladd,[3] Harris, Bell and Brunn,[4] Lewis[5] and Jackman[6] have made similar observations. The symptoms in these cases were those of an acute abdominal inflammation suggesting more or less appendicitis, while at operation an acute inflammatory engorgement of the distal segment of the ileum was found. The operative procedure varied between noninterference with the lesion and resection of the ileum, as for instance in Jackman's cases where the bowel wall was so severely compromised that it ruptured during the operation. In the few cases reported, the postoperative and final recovery has always been satisfactory.

The symptomatology of our cases was by no means uniform and hardly brings out any diagnostic feature which would facilitate the preoperative recognition of similar cases in the future. The findings in Case I were quite unusual and misleading. This patient, a five year old boy, presented all the symptoms of an acute intussusception: palpable tumor, visible peristalsis, intestinal hemorrhage and absence of temperature elevation. The considerable hemorrhage in this case is probably best explained as due to multiple erosions of the mucosa, which especially occur where submucous lymphatic plaques break down. At least the participation of the lymphatic apparatus in the inflammatory reactions was very conspicuous in the mesenteric lymph nodes, which attained a size usually seen only in acute mesenteric lymphadenitis. No palpable lymph nodes were present in the other two cases. Jackman, Lewis, and Ladd also fail to mention any lymph node hyperplasia in their acute cases, while all descriptions of chronic regional ileitis emphasize this finding.

The intensity of the inflammatory process and its sequelae varied considerably in our three cases. The inflammatory swelling was least pronounced in the last case and was commensurate with the comparatively mild and subacute symptoms. In the first case the engorgement of the ileum produced an intestinal obstruction, the swelling probably being intensified by exaggerated lymphatic reaction in the submucosa of the infantile gut. The severe infection of the bowel wall in the second case was accompanied by a localized peritonitis, but did not produce an obstruction.

Disease processes of the type here discussed are recent additions to the group of nonspecific inflammations of the intestinal wall. In this category may be included, at least until more is known about the pathogenesis of the different types, inflammatory processes of varying intensity, destructiveness and duration, but which have certain features in common: they are regional; *i.e.,* limited to a comparatively short segment of the intestinal tract. In contradistinction to the more common inflammations of the mucosa, they markedly involve the deeper layers of the intestinal wall. These inflammations evidently develop on the basis of a local infection and may run the course of an acute phlegmon or a chronic granuloma or any type of inflammation between these extremes. The bacteriologic study has only revealed bacteria of the common pyogenic or of the endogenous intestinal variety in the more

acutely inflamed tissues, while in the chronic granulating processes microorganisms may be practically absent. Trauma, various specific infections and malignant ulcerations of the mucosa and embolic infection, which occasionally lead secondarily to similar pathologic changes,[7] can be excluded; and one has to assume that etiologic factors of an unknown nature must cause the invasion of the bowel segment by organisms which ordinarily remain innocuous in the intestinal tract. The primary regional inflammations are not common in the intestinal tract, although phlegmonous infections are frequent in the vermiform appendix and intestinal diverticula. Here stagnation of contents evidently favors infection.

The chronic nonspecific granulomatous inflammations of the colon have been recognized for many years.[8, 9] It is now the opinion of most observers that chronic regional ileitis is essentially the same pathologic process, differing only in location. This contention is supported by observations of Colp,[10] Brown, Bargen and Weber,[11] Röpke[12] and Phillips,[13] who found terminal ileitis accompanied by a localized inflammatory process in the cecum or identical pathologic changes occurring in other segments of the ileum or even in the jejunum.

The acute, fulminant and phlegmonous infections, on the other hand, are usually found in the upper reaches of the intestinal tract, especially in stomach and duodenum. While phlegmons and mural abscesses have been also encountered in the colon, they take here, as Szabo[14] has pointed out, usually a less acute course and quite frequently subside into a chronic secondary stage.

Thus it is suggested that the character of the inflammation is to some extent influenced by its location at the various levels of the intestinal tract. In a general way, regional ileitis bears out this rule. In accordance with the location of this segment midway between the proximal and distal halves of the intestines, the inflammatory processes frequently take a chronic course. But acute processes are evidently just as frequent, yet do not, as a rule, progress as readily to suppuration and necrosis as the phlegmons of the stomach, duodenum and jejunum.

Judging from our own experience and from the increasing number of reports in the literature, the terminal ileum seems to be a site of predilection for regional inflammations. This greater frequency of infections in this segment of the bowels may possibly be accounted for by its functional and anatomic peculiarities. Normally, the flow of the intestinal contents is retarded at the ileocecal valve which necessarily leads to some degree of stagnation in the terminal ileum. Furthermore, the lymphatic tissue in the mucosa and submucosa is here more abundant than in other segments of the bowel, a factor which favors the absorption of microorganisms.

CONCLUSIONS

(1) Three cases of acute regional and intramural inflammation of the terminal ileum with symptoms of an acute intra-abdominal condition are described.

(2) The patients recovered after laparotomy without resection of the diseased segment of bowel.

(3) The relation of this disease to other nonspecific inflammations of the intestinal wall are briefly discussed.

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UNUSUAL INFLAMMATORY LESIONS OF THE ILEOCECAL REGION

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THE purpose of this paper is to call attention, by the use of three clinical reports, to the interrelationship of inflammatory lesions of the terminal ileum, appendix, cecum, and ileocecal lymph nodes. The first case is one of diffuse inflammation of the terminal ileum, appendix, and cecum with secondary involvement of the ileocolic nodes in the mesentery. In the second case, the tip of an old, inflamed appendix, obviously the primary focus of infection, was adherent to a mass of enlarged mesenteric nodes. The third case is one of acute primary typhlitis with localized, ulcerative, inflammatory changes in the wall of the cecum, and no involvement of the appendix.

CASE REPORTS

CASE I.—(Hospital No. 3326.) A white boy, aged 12 years, was admitted to the hospital because of pain in the right lower abdominal quadrant. The past and family histories were irrelevant.

The present illness began five days previously with cramp-like, generalized, abdominal pain associated with nausea but no vomiting. Examination two days later showed some congestion of the nasal mucous membrane and the rhinopharynx, slight tenderness to deep pressure throughout the lower abdomen, but no rigidity. The temperature was 99.4° F. by mouth, pulse 60. The following day the patient reported the passage of several tarry stools; the pain became localized in the right lower quadrant and persisted, with nausea and fever, until admission to the hospital.

The physical examination revealed a tall, thin, somewhat undernourished boy, with generalized cervical, axillary, and inguinal adenopathy of moderate degree. A hard, tender, nodular mass, with overlying muscle spasm, was palpable in the right lower abdominal quadrant, but could not be felt by rectal examination.

Clinical Pathologic Data.—Temperature 101.2° F., pulse 94, respirations 24. The leukocytic count was 11,920 with 69 per cent polymorphonuclear neutrophils. The urine was normal. The Wassermann was negative. The following day the leukocytic count was 13,300 with 76 per cent neutrophils.

Roentgenogram of the abdomen showed no evidence of calcified nodes nor anything else unusual. There was considerable gas in the colon.

Preoperative Impression.—Appendiceal abscess.

Operation was performed under ether anesthesia 12 hours after admission. McBurney incision. The amount of free fluid in the peritoneal cavity was increased and the omentum was adherent to a hard, indurated, nodular mass of inflamed lymph nodes just medial to the proximal portion of the ascending colon, which extended upward in a rather elongated form toward the midline (Fig. 1). The cecum, appendix, and terminal ileum were diffusely thickened, inflamed, and injected. There was no obvious obstruction of the intestinal lumen and resection was not considered advisable. The appendix and one enlarged node in the mesentery, just proximal to the ileocecal valve, were removed and the abdomen was closed without drainage.

Postoperative Course.—Characterized by a persistent elevation in rectal temperature which averaged approximately 100° F. each evening. Smear of the peritoneal fluid showed no microorganisms and no pus; the culture was sterile. The tuberculin test was negative on three occasions. The leukocytic count returned to normal. Examinations of the stools for ova, parasites, and occult blood were negative. Roentgenogram of the chest showed nothing unusual. Barium enema flowed freely around to the cecum and disclosed no abnormality in the outline or behavior of the colon. Patient discharged on the eighteenth postoperative day. The abdominal mass was not palpable.

Pathologic Diagnoses.—Subacute appendicitis and necrosis of lymph node. One section through the tip of the appendix showed a considerable amount of acute inflammatory exudate in the lumen; polymorphonuclear cells were scattered at rare intervals through the stroma and one of the lymphoid follicles was filled with these cells. There were also many plasma cells in the mucosa; no inflammatory reaction was seen in any

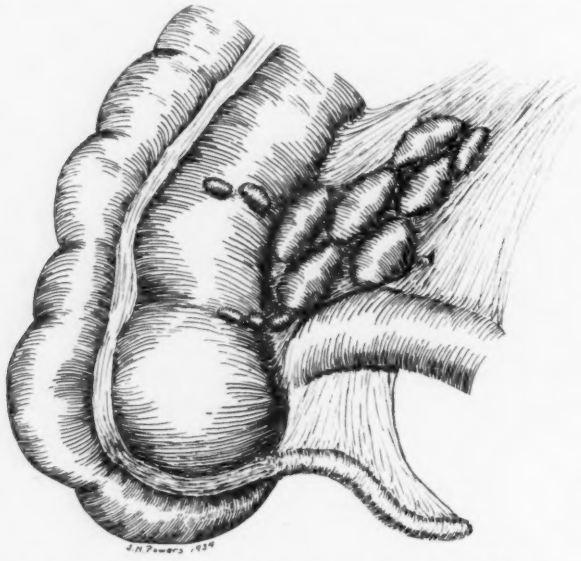

FIG. 1.—Diagrammatic sketch of the operative findings in Case I.

of the other coats. The section of lymph node was so necrotic that no cellular structure could be made out.

Comment.—The exact sequence of events in this case is by no means obvious. The fact that the patient has been completely cured by removal of the appendix (in view of the negative tuberculin and the absence of any microscopic evidence of tuberculosis in the lymph nodes), suggests that the process was a diffuse inflammatory one, primary in the appendix and secondarily involving the terminal ileum, cecum, and ileocecal lymph nodes. The microscopic examination of the appendix is not incompatible with this opinion. To be sure, the regional lymph nodes are not usually involved in the ordinary case of acute appendicitis, but such is also true with the common type of sore throat or infected finger. Yet one is not surprised occasionally to see diffuse edema of the hand with extensive involvement of the axillary nodes from an apparently trivial infection of the finger, and the author is inclined to believe a

similar type of reaction may rarely occur about the appendix, the ileocecal region, and the lymphatic structures which drain this portion of the alimentary canal.

CASE II.—(Hospital No. 8011.) A white male, 17 years of age, was admitted to the hospital because of abdominal pain and a tender swelling in the right side below the umbilicus.

The family history was irrelevant. The past history revealed an attack three months previously, similar to the present one, which lasted for two days and was followed by complete recovery. His weight was normal and his general health was good.

The present illness began six days prior to admission with a dull ache in the lower abdomen, anorexia, but no nausea or vomiting. Two days later castor oil was administered; the pain increased in severity and became localized in the right lower quadrant where the patient himself discovered a tender mass. Frequency of urination and nocturia ensued.

The physical examination was normal except for the abdomen, which was somewhat doughy and resistant throughout, with definite spasm in the right lower quadrant where an elliptical, tender, indurated, nodular mass could be felt in the right iliac fossa. The tumor extended from the umbilicus above to the pubis below and from the border of the right rectus muscle laterally to just beyond the midline; it was slightly movable. There were no palpable nodes in the axillae or groins.

Clinical Pathologic Data.—On admission the rectal temperature was 101.2° F., pulse 98. Leukocytic count 11,600 with 75 per cent polymorphonuclear neutrophils. The following day the temperature had risen to 102.6° F., pulse 80. Hemoglobin 76 per cent, red cells 3,788,000, leukocytic count 9,400 with 81 per cent neutrophils. The Wassermann reaction was negative in both antigens. Urine normal.

Roentgenogram of the abdomen showed nothing remarkable. Fluoroscopic examination of the colon, following the injection of barium, disclosed no evidence of pathology.

Preoperative Impression.—Appendiceal abscess lying directly beneath the anterior abdominal parietes.

Operation was performed under ether anesthesia 36 hours after admission. The abdomen was opened through a low right rectus incision, placed directly over the mass, to which the omentum was adherent by a few fresh inflammatory adhesions. The tumor consisted of numerous indurated, rounded, and egg-shaped lymph nodes varying from 1 to 3 cm. in length and 1 to 2 cm. in diameter which lay along the medial wall of the ascending colon and extended posteriorly into the retroperitoneal space. The nodes were inflamed, edematous, and matted together. The cecum and ascending colon were not constricted. The walls were thickened where they came in contact with the lymph nodes, and the anterior surface was inflamed and speckled in appearance although no actual tubercles were seen. A search was made for the appendix which was finally found as a thin, cord-like structure, extending upward behind the cecum and densely adherent to its posterior wall. It was removed in retrograde fashion from the base toward the tip which was firmly embedded in the largest of the lymph nodes (Fig. 2).

One node was sent to the laboratory for immediate microscopic examination which was reported to show no evidence of malignancy or tuberculosis. The abdomen was closed without drainage.

Postoperative Course.—Uneventful. Discharged 13 days postoperative. Examination at that time showed a marked decrease in the size and tenderness of the abdominal mass. The patient was free from pain or discomfort, his appetite was good and his bowels were regular.

Pathologic Report.—Acute adenitis of cecal lymph node; the node was heavily infiltrated with polymorphonuclear leukocytes. The appendix was 6 cm. in length

and varied between 2 and 5 mm. in diameter. A probe could not be inserted into the lumen and the histology was so distorted as to be unrecognizable.

The patient was seen one year later and stated that he had had some residual soreness in the right lower abdominal quadrant for one month following his discharge from the hospital but none since. Examination showed a well healed wound just lateral to which could be palpated a small firm, nodular, nontender tumor. The leukocytic and differential counts were normal. A year later the abdominal mass had disappeared.

Comment.—This case is unquestionably one of acute mesenteric adenitis secondary to appendicitis. On the basis of the operative findings and the microscopic appearance of the tissue, it seems probable that the nodes were involved primarily by direct extension of an inflammatory process in the appendix which completely destroyed the usual histology of this organ during

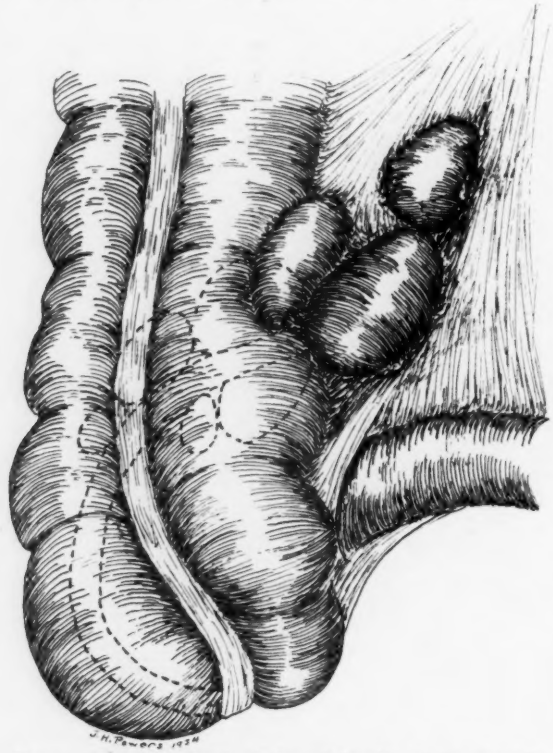

FIG. 2.—Operative sketch of the relationship of the appendix to the ileocecal lymph nodes in Case II.

the initial illness, and that the second attack of pain was due to peritoneal irritation by the mass of inflamed lymph nodes rather than to the recurrence of appendiceal colic.

CASE III.—(Hospital No. 6011). A previously healthy man, 33 years of age, a painter by occupation, entered the hospital because of abdominal pain. The past and familial histories were irrelevant.

The present illness began four days previously with severe cramplike abdominal pain, located chiefly in the left upper quadrant but radiating across to the right side. There was no nausea or vomiting. The following day the pain was largely relieved by the local application of ice to the abdominal wall; after removal of the ice packs the

cramps recurred at half-hourly intervals until admission to the hospital two days later. The patient had little desire for food; his bowels moved satisfactorily without catharsis and the stools were normal in character.

The physical examination revealed no lead line. The heart and lungs were normal. The abdomen was tender to pressure throughout the right side, particularly in the lower quadrant where overlying muscle spasm and cutaneous hyperesthesia were present. Rectal examination disclosed nothing unusual.

Clinical Pathologic Data.—Temperature 100.2° F., pulse 94 and respirations 20. The leukocytic count was 9,700 with 71 per cent polymorphonuclear neutrophils. The urine was normal and the Wassermann reaction was negative.

Preoperative Impression.—Acute appendicitis.

Operation was performed under general anesthesia. The abdomen was opened

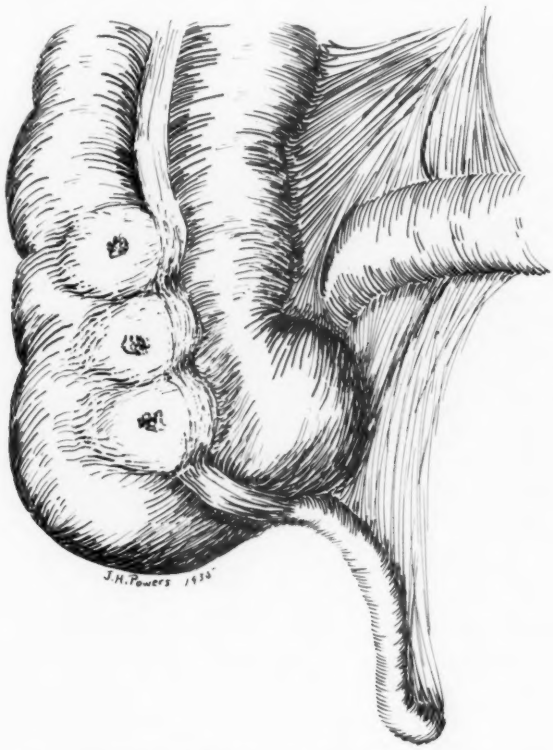

FIG. 3.—(Case III.) Primary ulceration of the cecum.

through a right rectus incision. Fresh inflammatory adhesions were found about the cecum; the appendix was congested but no fibrin was present. Three cm. above the base of the appendix the serosa of the cecum was acutely inflamed and granular in appearance. Palpation of this area disclosed three definite nodules in the cecal wall, about 2 cm. in thickness and 1 cm. apart (Fig. 3). No definite craters could be made out. The appendix was removed, the inflamed portion of the cecum resected and the wound closed in layers with silk.

Postoperative Course.—Characterized by an abrupt elevation of temperature to 102.2° F. which gradually subsided to normal on the sixth day. The wound healed satisfactorily and the patient was discharged at the end of two weeks. There has been no subsequent complaint.

Pathologic Diagnosis.—Ulcers of cecum and normal appendix. The ulcers in the excised cecal wall were 2.5 by 1 cm. in diameter and oval in shape, with their long axes transverse to the lumen of the gut. The surrounding intestinal wall was thickened and injected and the serosa was inflamed. The surface of the ulcers was covered with necrotic exudate beneath which could be seen microscopically a dense layer of polymorphonuclear leukocytes. Lying on the muscularis was a still deeper layer of inflammatory cells, fibroblasts and fibrin. Sections stained with the Gram stain showed numerous gram-positive bacilli, particularly abundant in the superficial necrotic membrance covering the ulcers. Sections of the appendix revealed nothing abnormal.

Comment.—This case is doubtless one of primary nonspecific typhlitis. The appendix was not involved in the inflammatory process which was strictly confined to the cecum. Several similar cases have been reported in the past, either as "stercoral ulcers," simple inflammatory lesions, or ligneous infection of the cecum.

Discussion.—Great diversity of opinion exists regarding the etiology of these unusual inflammatory lesions of the terminal ileum, cecum, and appendix, their relationship to one another, and to acute mesenteric adenitis along the paths of lymphatic drainage. A large series of cases may be found in the literature under the general term of benign, nonspecific granulomata. This group includes both acute and chronic inflammations of the small and large intestine, the cause of which remains unknown.

Regional Ileitis.—In 1932, Crohn, Ginzburg, and Oppenheimer[1] attempted to segregate from this heterogenous collection a group of inflammatory lesions of the terminal ileum to which the term "regional ileitis" was applied. They described the condition as a definite clinical and pathologic entity which did not extend beyond the ileocecal valve and bore no relation to the appendix except such involvement as might occur to the outer coats of that organ by the presence of adjacent inflammatory disease.

In 1933, Harris, Bell, and Brunn[2] published three cases, in one of which the jejunum was mainly involved; the adjacent mesentery in each instance was thickened and edematous and the lymph nodes were enlarged. Because of the involvement of the small intestine above the terminal ileum the name "chronic cicatrizing enteritis" was suggested as a more appropriate term. In a later report, Crohn[3] records a case in which the whole ileum and lower jejunum were involved and mentions another, "probably tuberculous," of diffuse involvement of the lower jejunum and upper ileum. This same authority, in his discussion of Kantor's paper,[4] stated that the disease was much more common than hitherto suspected and mentioned one case in which the inflammatory area transcended the ileocecal valve to involve the cecum. These facts tend to support the opinion of Homans and Hass[5] who presented two cases with a similar clinical picture. They refused to admit that the malady was a specific pathologic entity and considered a possible but unproved relationship of the disease to appendicitis. The appendix in one of their cases was ulcerated and in the other was partly destroyed by fibrosis. In both instances the mesenteric lymph nodes at the ileocolic angle were numerous,

enlarged, and inflamed. Clute[6] has published two such cases, in each of which the mesentery was boggy, edematous, and filled with hyperplastic lymph nodes which he suggested might be the primary site of the infection. Rockey[7] reported four cases of thickening of the terminal ileum with mesenteric adenitis in children, in none of which the appendix was involved either grossly or microscopically. All the patients were cured by simple appendicectomy, a fact which seems strange if this organ were not the primary focus of infection.

From this brief résumé of the literature it is apparent that the etiology of nonspecific inflammation of the terminal ileum is entirely obscure, that the pathologic process may involve other portions of the small intestine and at times may even transcend the ileocecal valve to include the proximal portion of the large bowel.

Inflammatory Lesions of the Cecum.—In reviewing cases of "typhlitis" and nonspecific inflammatory lesions of the cecum one is impressed by the overwhelming number in which the appendix is implicated either by previous or coexistent inflammation. In the latter type, the organ is involved in the general process and is assumed, with some degree of reason, to be primarily responsible for the disease. In the former a history is obtained of previous appendicectomy or of recurrent attacks of abdominal pain which might be clinically ascribed to the appendix. And often when the organ appears grossly normal at operation, microscopic examination reveals evidence of chronic appendicitis. Frequently the ileocolic lymph nodes are described as more numerous than normal, enlarged, juicy, hyperplastic or inflamed.

On the other hand, simple inflammatory tumors of the transverse colon and sigmoid have been reported, so one may reasonably assume that a similar type of lesion occurs in the cecum which has no primary relationship to the appendix. Bernard and Melone,[8] in summarizing an article on acute typhlitis, state that unquestionably such a disease does exist but that the limits of primary typhlitis ought to be more carefully defined for much that has been described as such is, in reality, secondary to appendicitis.

Wilkie,[9] in his studies on the pathology of the cecocolic sphincteric tract of the colon, suggests a possible explanation for the susceptibility of this part of the intestinal tract to morbid processes. He calls attention to the work of Keith in demonstrating three distinct segments in the proximal part of the colon of air breathing vertebrates, namely, the cecum, the cecal colon, and the cecocolic sphincteric tract. This tract, immediately distal to the caput coli, is frequently in a state of tonic contraction in herbivorous animals and corresponds anatomically to a contracted zone in the human colon which is often described at the sphincter of Busi.[10] Furthermore, this sphincteric region coincides with that in which pathologic processes are particularly prone to occur. Roith and others have established that anastalsis takes place in the proximal part of the human colon, and radiologic studies, after the ingestion of an opaque meal, have demonstrated repeatedly that the forward passage of the intestinal contents is delayed for several hours in the cecum, a delay

which is due not so much to a passive obstruction as to an active repulsion of these contents until cecal digestion and absorption have been completed.

Wilkie concludes: "All writers are agreed that the human cecum approaches more nearly to the herbivorous than to the carnivorous type. As Keith points out, the diet of highly civilized races is so different from the crude and primitive food supply for which the human cecum was originally evolved that it is not surprising to find that this portion of the alimentary canal, working as it does under unnatural conditions, should be especially liable to morbid changes."

Furthermore, pathologic lesions are prone to occur in other parts of the gastro-intestinal tract just at or proximal to points of constriction. The predilection of peptic ulcers for the region of the pylorus is well known. If temporary stasis at this point contributes toward ulceration of the stomach, it is reasonable to expect that ulcers might also occur in the cecum (as in fact they actually do) just proximal to the cecocolic sphincteric tract. To pursue the theory further, the decrease in the lumen of the terminal ileum and the constriction at the ileocecal valve may be at least partially responsible for the development of regional ileitis. Further support for this hypothesis is suggested by the fact that such pathologic processes frequently undergo spontaneous resolution if an anastomosis be performed around the obstructed and inflamed portions of intestine.

Ileocecal Lymphadenitis.—The etiology of inflammatory lesions of the lymph nodes situated in the ileocecal angle of the mesentery is still a subject of controversy. Many authors believe that the majority of these cases is tuberculous in origin, even when no tubercles or bacteria are demonstrable by microscopic examination. On this basis, simple lymphoid hyperplasia is explained as due to "absorption of toxins from ulcerated tuberculous lesions in the musoca" or "reactions provoked by invasion of the tubercle bacillus,[11, 12] possibly from the irritation of toxins." Unfortunately, sound surgical judgment at the operating table has not permitted exploration of the lumen of that portion of the intestine which was suspected of being the primary tuberculous focus. Unquestionably, many cases of mesenteric lymphadenitis are tuberculous in origin; on the other hand, the absence of histologic evidence of this disease in the excised nodes, negative intradermal tests, the immediate disappearance of symptoms, and rapid healing in many cases suggest a simple pyogenic origin. Wilensky[13, 14] has taken a firm stand on this basis and contributed much toward clarifying the etiologic and pathologic confusion. According to his classification, mesenteric adenitis may be divided into simple, suppurative, tuberculous, and calcified. The first includes a uniform discrete enlargement of the mesenteric nodes in which the pathologic picture is one of simple hyperplasia. The second differs only in degree; more extensive pathologic changes and suppuration occur. The third is frankly tuberculous and the fourth is a terminal affair, the end result when healing occurs in either the second or third groups. Abundant evidence to support the existence of simple hyperplastic or pyogenic adenitis may be found in the reported cases

of regional ileitis where the involvement of the mesenteric nodes is apparently secondary to an inflammatory process in the ileum. That such an adenitis is not usually found in the ordinary case of acute appendicitis may be due either to the fact that the patient is operated on so early that sufficient time has not lapsed for the ileocecal nodes to become involved, or, in the event of a late operation, to the fact that the efforts of the surgeon to avoid contamination of clean peritoneal surfaces do not permit exposure and exploration of the mesentery.

From the standpoint of anatomic knowledge, one might expect frequent involvement of the ileocolic nodes in the presence of appendiceal inflammation. Kelly[15] states: The lymphatics of the appendix may be divided into three more or less distinct systems which are identified with the coats of the appendix,

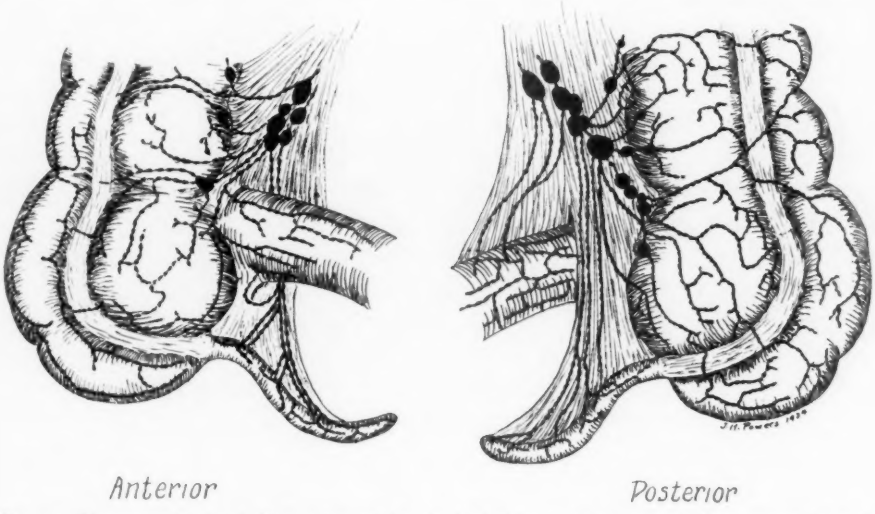

FIG. 4.—Diagrammatic sketch illustrating the lymphatic drainage of the terminal ileum, appendix, and cecum (after Kelly).

the superficial, lying in the serosa; the middle, situated between the submucosa and layer of circular muscle fibers; and the deep, consisting of a very complex anastomosing network, the central layer of which lies between the mucosa and the muscularis mucosae; delicate, fingerlike terminal branches, running parallel to and between the glands of Lieberkühn, drain the mucous membrane. The collecting channels, running in pairs, form one to three separate bundles passing along the free border of the meso-appendix. If there are more than one, the others run at fairly regular intervals between the main bundle and the cecum. All converge toward the appendiceal nodes which are generally situated at the lower median angle of the ileocolic chain. Glandular substations, or isolated glands in the meso-appendix or in the appendicocecal angle, are rare (Fig. 4). In view of (*a*) the similarity between the lymphatic apparatus of the appendix and terminal ileum; (*b*) the frequency of mesenteric adenitis as an accompaniment of regional ileitis; and (*c*) the lack of interest in the ileocolic nodes

in the presence of obvious appendicitis, it is quite possible that these nodes are involved more frequently than is generally suspected.

In his first paper, Wilensky[13] felt that the source of infection in cases of ileocecal lymphadenitis was most commonly in the appendix. In the second paper,[14] six years later, he believed that the Peyer's patches in the intestinal wall formed the point of entry similar to the rôle which the tonsils play in cervical adenitis—and that no clinical relationship existed between appendicular infection and mesenteric adenitis. The prompt subsidence of symptoms following appendicectomy in his early cases, in the cases reported by Rockey,[7] and in the first two cases recorded above, and the anatomic relationship of the lymphatic channels of the appendix to the ileocecal lymph nodes suggest that the appendix does play some part in the etiology of this interesting form of glandular inflammation.

SUMMARY

(1) Three cases of unusual inflammatory lesions of the ileocecal region have been described in detail. The first was that of a patient with diffuse inflammation of the terminal ileum, appendix, and cecum, and secondary involvement of the ileocolic nodes in the mesentery; in the second case, the tip of an inflamed appendix was adherent to a mass of enlarged mesenteric nodes; the third case was one of primary typhlitis with localized, ulcerative, inflammatory changes in the wall of the cecum.

(2) Regional ileitis, acute nonspecific inflammation of the cecum, and mesenteric lymphadenitis in the ileocolic angle have been discussed.

(3) Certain etiologic factors and the possible interrelationship of these lesions to one another have been suggested.

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PRIMARY CARCINOMA OF THE FALLOPIAN TUBES

REPORT OF TWO CASES

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IN A thorough search of the American and foreign literature from 1888 to 1935 on primary carcinoma of the fallopian tubes, 323 cases were found to have been reported. This is a far greater number than is mentioned in the American literature. Of Liang's[1] 214 recorded cases in 1926, 107 were reported in the German literature. Only brief mention is made of the subject in text-books of pathology and surgery. Even this number of cases is too small to consider the subject of primary carcinoma of the fallopian tubes as complete; hence the reason for reporting two additional cases. Many more will have to be reported before we can gather enough facts about this subject to class it as a definite entity among gynecologic diseases.

Today so little is known about the subject by most surgeons that the possibility of the diagnosis is seldom considered—none did in the past with the exception of Falk,[2] and this only after removing a piece of tissue through an abdominal puncture.

Orthmann[3] was the first to describe primary carcinoma of the fallopian tubes in 1888. In 1895, Sänger and Barth[4] gave a detailed description and classification of primary carcinoma of the fallopian tubes, declaring that it always develops at the seat of a chronic and most frequently suppurative salpingitis long quiescent. Peham,[5] in 1903, collected 63 cases. Three years later Orthmann[6] collected 84 cases, and in 1910 Doran[7] tabulated in detail 100 cases. A year later Andruze-Acher[8] added 15 and in 1914 Vest[9] 32. In 1925, Liang collected 214 cases. Wechsler,[10] in 1926, tabulated 192 cases from the literature and reported four new cases. Wharton and Krock,[11] in 1929, found in the literature 230 cases and reported 14 of their own from Johns Hopkins and neighboring hospitals. When Johnson and Miller[12] reported their case in 1931, they found in the literature 250 cases. Walther Haupt,[13] in 1933, collected 321 cases and reported one of his own. Kahn and Norris[14] reported a case in 1934, making a total of 323 cases of primary carcinoma of the fallopian tubes from 1888 to 1934.

Among 2,020 gynecologic specimens at the University of Pennsylvania, only one case of primary carcinoma of the fallopian tubes was found (Norris[15]). Only five cases were found in 35,000 patients with gynecologic conditions at the Johns Hopkins Hospital (Wharton and Krock); at the Lenox Hill Hospital four times in 5,870 specimens (Wechsler); at Bellevue Hospital three times in 30,000 patients with gynecologic conditions (Barrows[16]). According to Johnson and Miller[12] it occurs in about 0.45 per cent of all genital tumors.

Carcinoma of the fallopian tubes occurs most frequently about the menopause, usually between 40 and 50, but cases have been observed as early as 18 (Johnson and Miller), and as late as 73 (Mentul, quoted by Wechsler). Pelvic inflammation and sterility, in turn due to pelvic inflammation, are predisposing factors (Sänger and Barth, Norris, Ewing,[17] Doran). The importance of pelvic inflammation is questioned by Vest, Wechsler and Liang. Some cases are on record that had no history of inflammation. One case is reported that occurred in a virgin (Spencer[18]). The second case is being reported in this paper. The majority of cases begin as multiple papillary outgrowths of an inflamed mucous membrane which become malignant (Ewing, Doran). Over 75 per cent were reported as papillary carcinoma. The classification of Sänger and Barth seems to be the most logical and simple. They occur either as papillary carcinoma or papillary alveolar carcinoma. The latter is sometimes reported as adenocarcinoma.

The symptoms of pain, serosanguineous discharge and palpable tumor are not typical of this disease alone, especially when one takes into consideration that about 9 per cent of cases are associated with uterine fibroids, ten per cent with cystic tumors of the ovaries, and chronic adnexal diseases, for which so many of these cases are being operated upon.

The pain differs from that of carcinoma of the uterus in appearing early in the disease. It is a constant, lancinating, colicky pain, which in some cases stops abruptly following a profuse vaginal discharge. This occurs when a distended tube empties itself through the uterine ostium, as reported by Roberts[19] and Edelberg.[20] Occasionally, some malignant tissue is extruded with the discharge. When the abdominal ostium is open, the contents of the tube are emptied into the abdominal cavity, thus disseminating the malignancy to the peritoneum, followed later by ascites. Ascites, however, occurs only in about 10 per cent of the cases, as in the majority the abdominal ostium is closed. In lymphatic metastasis ascites may follow in the same manner through peritoneal and mesenteric involvement.

In most obscure surgical cases, diagnosis is usually made in the operating room. Even this is not always the case in primary carcinoma of the fallopian tubes, unless the tube is sectioned and examined before the abdomen is closed. Wharton and Krock called attention to this fact. The fear of criticism by the pathologist for "spoiling a specimen" by sectioning often results in secondary operations after the pathologic report is received.

Diagnosis of primary carcinoma of the fallopian tubes may be made when the patient complains of a profuse serosanguineous discharge with various menstrual irregularities, abdominal pain with a palpable adnexal tumor, and a negative report of the uterine curettings. Indiscriminate use of radium to arrest vaginal bleeding without a pathologic diagnosis should be condemned, as some cases in the past have proved to be primary carcinoma of the fallopian tubes.

Radical surgery with the removal of both tubes and ovaries and complete hysterectomy with wide excision of the broad ligaments is the treatment of

choice. This is followed later by roentgen ray therapy. The operative mortality is about 6 per cent.

The prognosis of primary carcinoma of the fallopian tubes is very poor. Metastasis usually takes place by the lymphatic route, involving the retroperitoneal nodes, resulting in large tumor masses filling the abdomen, or by direct implantation from a tubal ostium involving the omentum and mesentery, associated with ascites in about 10 per cent of cases. Metastasis is usually limited to the lower abdomen. The liver was involved in only three cases. It is the most malignant type of all gynecologic tumors, perhaps including carcinoma of the ovaries. As stated by Wharton and Krock, "If we were to base the prognosis entirely on the results of the past, it would be necessary to conclude that the outlook is almost hopeless." Only 4.5 per cent of the cases reported have obtained a three year cure and but seven cases survived longer than three years. Haupt's case survived nearly 20 years; Fonyo's[21] 13 years; Zweifel's[22] eight years; Veit's[23] seven years; Jahkola's[24] six and seven years, and Benthin's[25] five years.

Case Reports

Case I.—E. K., a white adult female, aged 52, not married, was admitted to the Brooklyn Cancer Institute January 8, 1935. Chief complaint: lower abdominal pain and vaginal bleeding. The bleeding was of two weeks' duration, scanty and serosanguineous in character. The pain was intermittent. The patient had had her menopause two years previously. Menses began at 18 with only occasional irregularities, lasting five days. She had had only a slight amount of leukorrhea between menstrual periods, and never any pelvic pain before. She was a virgin. She had had the usual childhood diseases and had become deaf following measles. Her family history was irrelevant.

Physical examination revealed a white adult female, well nourished, appearing much younger than her age, weighing 118 pounds, about five feet three inches in height, temperature 99°, pulse 82, respirations 20, blood pressure 120/80. The abdomen was soft, ovoid and not tender. There were no visible or palpable masses present. Pelvic examination revealed a virginal introitus, admitting only one finger. A mass the size of a small orange was felt in the left fornix. The body of the uterus could not be palpated. Heart and lungs normal. Urine normal and blood Wassermann negative. Hemoglobin 60 per cent, red blood cells 3,700,000, with 4,950 white cells, 81 per cent polymorphonuclears, 19 per cent lymphocytes.

January 10 an exploratory laparotomy was done by Dr. John J. Gainey. The left tube and ovary were found to be buried by adhesions in the left fornix and culdesac, forming one mass. On freeing this a tumor the size of a small orange was found at the fimbriated end of the tube. A left salpingectomy and appendectomy were done. The patient made an uneventful recovery.

Pathologic report by Dr. Polayes: *Macroscopic Examination.*—Specimen an ovid mass 8½ cm. in length and 4 cm. in greatest diameter. The tissue consists of the fallopian tube, the distal end of which is markedly dilated and filled with gray granular friable tissue. There is a small cystic area about 1 cm. in diameter on the surface of the mass.

Microscopic Examination.—Tissue consists of tubal wall, the lining of which shows a transition into malignant epithelial cells which grow in papillary formation and destroy the architecture of the ampulla of the tube beyond recognition. Section through the tumor mass shows a solid growth of epithelial cells which are entirely anaplastic (Fig. 1). *Diagnosis:* Papillary adenocarcinoma of ampulla.

A week later the patient was given a transfusion of 500 cc. of blood and a

panhysterectomy was done, removing the left ovary, right tube and ovary and uterus.

Pathologic report by Dr. Polayes: *Macroscopic Examination.*—Specimen consists of two portions, one composed of uterus and its appendages, and the cervix uteri. The uterus measures 4½ by 4½ by 2 cm. The right tube measures 9½ cm. in length, 6 Mm. in its widest diameter, and the midportion of which presents a hemorrhagic infiltration involving the entire wall at this point for a distance of 3 cm. A small peritubal cyst is present. The left tube is missing and suture is present at the uterine cornu. Each ovary measures 2 cm. in longest diameter.

The left ovary contains a hemorrhagic area over the distal half. On section this hemorrhagic infiltration extends into the substance of the ovary for a distance of one-

FIG. 1.—(Case I.) Papillary adenocarcinoma of the fallopian tube (×100).

fifth Mm. The remaining portion is made up of round yellowish firm corpus luteum structure. The right ovary shows a cyst-like structure at one point, the lining of which is moist, red-pink and extends to the surface. The anterior wall of the endometrial cavity presents an irregular polypoidal appearance with the lining endometrium glistening in character. The polypoid areas are soft and cystic-like in consistency. The myometrium through this point is gray-pink in color and of normal, soft consistency. There is a small subserous pedunculated nodule over the posterior portion of the body which measures 5 Mm. in diameter.

Microscopic Examination.—Uterus: The endometrium is the seat of a marked glandular hyperplasia. Section through the left cornual region shows the presence of a number of endometrial gland inclusions within the muscular layer. Portions of the wall here are completely hyalinized, while others are necrotic and infiltrated with polymorphonuclears. This reaction is found in a fairly well circumscribed area of degenerating fibromyomatous structure. The right tube shows no changes other than flattening of the papillae and peritubal extravasation of blood.

Left ovary: There are numerous corpora albicantes and very marked hyalinization and sclerosis of the ovarian vessels. One portion contains follicular cysts, several of which show papillary cystadenomatous changes.

The right ovary shows changes similar to the left ovary. *Diagnosis:* (1) Glandular hyperplasia of endometrium. (2) Degenerating fibromyoma uteri. (3) Papillary cyst adenoma of ovaries, bilateral.

The patient made an uneventful recovery and was discharged February 10. She then received postoperative roentgen ray therapy consisting of 1,720 R units to the lower abdomen.

CASE II.—M. R., a white adult female, aged 39, was admitted to Prospect Heights Hospital December 1, 1931, complaining of lower abdominal pain and marked vaginal

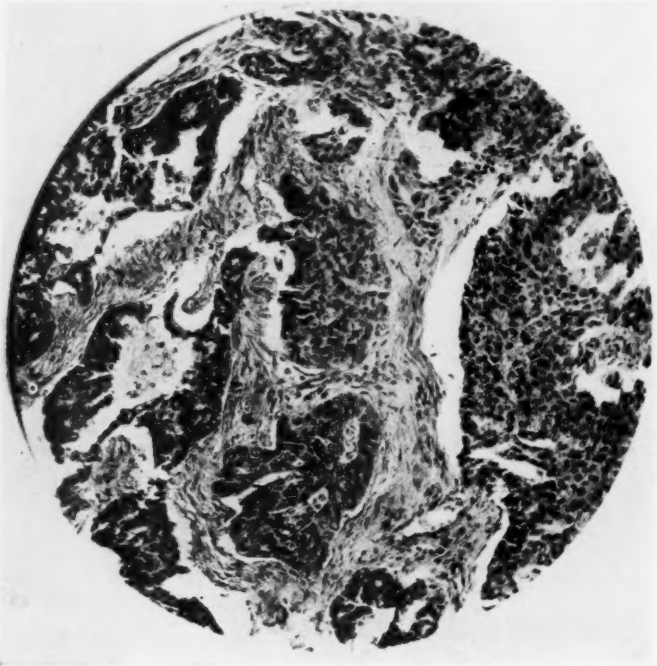

FIG. 2.—(Case II.) Papillary adenocarcinoma of the fallopian tube (×250).

discharge of four months' duration. Her menses began about the age of 14, were always regular. She had one child which died in infancy. No other pregnancy. Her last menstrual period was on November 27, 1931. In the past four months she had developed a marked mucopurulent discharge accompanied by lower abdominal pain, which failed to respond to any treatment. Her family and past history were irrelevant.

Physical examination revealed a white adult female, moderately developed, not acutely ill. Temperature 99°, pulse 80, respirations 20. Pelvic examination revealed the cervix slightly displaced to the left by a cystic mass in the right fornix intimately adherent to the body of the uterus, which was retroverted and extended about two fingers above the symphysis. There was a great deal of tenderness and pain in the right lower quadrant on bimanual examination; this was increased on manipulation of the cervix, which was slightly eroded, nodular, and exuded a mucopurulent odorless discharge. The rest of the physical examination did not reveal any pathologic findings. Heart and lungs normal.

The urine showed a slight amount of albumen, otherwise negative. Hemoglobin

72 per cent, red blood cells 3,780,000, white cells 10,000, 86 per cent polymorphonuclears and 14 per cent monocytes, 4 per cent band cells and color index 0.97.

December 3 she was operated upon by Doctor Gainey. The abdomen was opened through a low median incision and explored. Both tubes and ovaries were found intimately adherent to the uterus. The right tube was distended with fluid. Its fimbriated end contained a yellowish white solid mass about the size of a small orange, and was sealed up by the ovary, which was closely adherent to the ostium. The left tube was only slightly distended. Both ovaries were small and fibrocystic in character. The uterus was hard, firm, and covered with small fibroids. It was found on section that its lumen was almost completely obliterated by numerous similar small fibroids.

Both tubes and ovaries were removed, followed by a supracervical hysterectomy. The pathologic report of the right fallopian tube was primary adenocarcinoma (Fig. 2). There was no other malignancy found in the rest of the pelvic organs.

The patient made an uneventful recovery and was discharged December 15, two weeks postoperative. She was free of symptoms, had gained weight, and enjoyed her daily occupation. August 28, 1933 (two years and eight months after operation), she developed symptoms of acute intestinal obstruction. She was again operated upon and a band of adhesions was found about the ileocecal region. A thorough exploration did not reveal any evidence of recurrence or metastasis. The adhesions were freed and an ileostomy performed. She died September 10 (12 days after operation) from pulmonary embolism.

SUMMARY

(1) Three hundred twenty-three cases of primary carcinoma of the fallopian tubes were found in the literature.

(2) It occurs in about 0.45 per cent of all genital tumors.

(3) Chronic inflammation is believed to be a predisposing factor.

(4) Diagnosis is extremely difficult. Even at laparotomy diagnosis is often missed unless the tumor is sectioned by the surgeon.

(5) Symptoms of serosanguineous discharge with various menstrual disturbances and abdominal pain associated with a palpable adnexal tumor and a negative uterine curettage are important aids to the diagnosis.

(6) Radical surgery with the removal of both tubes and ovaries and a complete hysterectomy with wide excision of broad ligaments is the treatment of choice. This is to be followed later by roentgen ray therapy.

(7) Prognosis is very poor. Only seven cases survived longer than three years.

(8) Two cases of primary carcinoma of the fallopian tubes are reported. One occurred in a virgin. There is only one other similar case reported in the literature.

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FRACTURES OF THE HUMERUS

ANALYSIS OF TREATMENT AND RESULTS OF 200 FRACTURES

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IN PRESENTING the salient facts derived from a careful analysis of 200 successive cases of fractures of the humerus entering Harlem Hospital in the three years prior to November, 1933, on the service of the late Dr. John Fox Connors and his successor, Dr. Charles Cassasa, an effort has been made to avoid unnecessary statistics and to demonstrate that with the treatments advocated the hospitalization period is greatly reduced, the patient is made ambulatory in traction shortly after admission, and rarely is it necessary for an open operation to be performed in order to attain good anatomic and functional results.

TABLE I

TOTALS

200 consecutive cases of fracture of the humerus in three years.
16 deaths (all due to causes other than the fracture alone).
9 A. O. R. (left hospital on own responsibility).
8 transfers (to Psychopathic and Prison Wards at Bellevue Hospital).
167 cases treated.
106 cases returned for follow up in clinics.
90 per cent satisfactory anatomic results.
89 per cent satisfactory functional results.

Of these 200 cases entering the hospital only 167 remained long enough to be treated. Of the remaining 33 who were discharged without complete treatment, eight were transferred to another hospital, and nine left the hospital at their own request. The remaining 16 cases died, but in every case the cause of death was from causes other than the fractured humerus, *i.e.*, a fractured skull, or shock, resulting from such varying causes as abdominal evisceration, extensive severe burns, multiple fractures or chronic cardiac disease.

TABLE II

ARM, AGE AND SEX INCIDENCE

Arm	Left humerus fractured in 122 cases (61 per cent)	
	Right humerus fractured in 78 cases (39 per cent)	
Females fractured left arm more frequently than males (62 per cent)		
Sex	Males fractured arms in 121 cases (60½ per cent)	
	Females fractured arms in 79 cases (39½ per cent)	
Age	21 per cent (42 cases) were in children under 15 years of age.	
	52 per cent (104 cases) were in the working age group	(15 to 50 years).
	10 per cent (20 cases) were in the less active age	(50 to 60 years).
	17 per cent (34 cases) were in the elderly group	(over 60 years).

The most frequent causative factor in these fatal cases was a fall from a high window.

Side Incidence.—The left humerus is fractured much more frequently than the right in spite of the dexterity of the major part of the population. This condition has been a constant factor during each year the statistics have been analyzed, and may be due to the fact that an individual invariably thrusts his right or stronger arm forward to ward off an impending danger. The impact of the force does not injure this extended arm but causes the person's body to be thrown by the force, and the opposite arm which is not braced to protect itself is crushed against the ground or some other hard and stable object. It is felt that only a small percentage of these fractures of the humerus were caused by direct trauma from the initial force. Sixty-one per cent (122 of the 200 cases) of the fractures were in the left arm and only 39 per cent in the right arm, and 11 of the 12 compound fractures were in the left arm.

Sex Incidence.—As to the incidence according to sex, it is perhaps quite natural that men should have comprised 60½ per cent of the cases and the females the remaining 39½ per cent. Females fractured their left arms slightly more frequently than did the males.

Age Incidence.—With regard to the age incidence, 52 per cent of the cases were in people of the working age (namely, between 15 and 50 years), 21 per cent were in children, 17 per cent in people between 50 and 60 years and naturally less active, while only 10 per cent were in people over 60 years of age.

Site of Fracture.—The location or site of fracture, strangely enough, varies with the age of the patient, for the older the patient the higher the level of the fracture, and inversely the younger the patient the lower is the usual level of the site of the fracture. Fractures below the upper third of the humerus are relatively few in people older than the working age. Fractures of the shaft form 58 per cent of these fractures in people of the working age, while the most common arm injury in children is a supracondylar fracture.

TABLE III

LOCATION OF THE FRACTURE (SITE)

Head (alone)	1
Head and surgical neck	3
Surgical neck or anatomic neck	37
Surgical neck and greater tuberosity	15
Surgical neck and upper shaft	2
Surgical neck and supracondylar	1
Greater tuberosity	13
Greater tuberosity and upper shaft	2
Upper third of shaft	14
Middle third of shaft	60
Lower third of shaft	16
Supracondylar	29
Internal condyle	4
External condyle	3

TABLE IV

LOCATION OF THE FRACTURE (PERCENTAGE)

Fractures of the head of the humerus	4 (2	per cent)
Fractures of the surgical neck of the humerus	57 (28½	per cent)
Fractures of the greater tuberosity of the humerus	30 (15	per cent)
Fractures of the upper extremity of the humerus	73 (36½	per cent)
Fractures of the upper third of the humerus	87 (43½	per cent)
Fractures of the middle third of the humerus	60 (30	per cent)
Fractures of the lower third of the humerus	53 (26½	per cent)
Fractures of the lower extremity of the humerus	37 (18½	per cent)
Fractures of the shaft of the humerus	92 (46	per cent)

TABLE V

LOCATION OF THE FRACTURE (ANALYSIS)

(A) The site of fracture tends to be higher in the bone the older the patient, and, inversely, lower in the bone the younger the patient.
(B) Fractures of the shaft are most common in persons of working age, forming 58 per cent of this group of cases.
(C) Fractures of the upper third are most common over 50 years of age.
(D) Fractures of and above the condyles are most common in children and least common in the elderly.

Complications.—Complications of these are of two main types, direct complications, pertaining to the fractured humerus itself, and indirect complications, referring to injuries and fractures of other parts of the body, or severe medical ailments or diseases.

TABLE VI

309 COMPLICATIONS OF FRACTURES OF THE HUMERUS

85 Direct Complications

21 compound fractures (12 of the 21 treated).
29 intra-articular fractures.
12 dislocations of the shoulder.
7 pathologic fractures.
16 nerve injuries (radial 13, ulnar 2, median 1).

224 Indirect Complications

171 additional bones fractured.
53 internal or cerebral injuries.
46 cases were uncomplicated.
47 cases had direct and indirect complications.
154 were complicated.

The direct complications numbered 85 in all, comprising 21 cases of compound fractures (10½ per cent); seven were pathologic fractures (3½ per cent); 29 intra-articular fractures; while 12 cases had an associated dislocation of the head of the bone (6 per cent), and 16 cases caused a resultant nerve injury (8 per cent).

(A) Of the compound fractures 12 remained to be treated and of these 12 cases only one case became infected and 11 healed cleanly. Of the

21 compound fractures, seven died on admission from other or multiple causes, two left the hospital either on their own responsibility or on transfer, and 12 remained to be treated (11 were in left arm and 11 in males). Seventy-five per cent had good anatomic results and 78 per cent had good functional results. No case of compound fracture of the humerus who survived his other injuries was admitted to Harlem Hospital between October, 1931, and November, 1933. This is significant, since it was at the beginning of this time that an order to all ambulance surgeons to apply traction in a Thomas splint to all fracture cases before transporting them was rigidly enforced. This application of emergency traction in a Thomas splint has prevented many complications, in addition to making the patient more comfortable while being transported, and the reduction of the fracture more simple by preventing overriding and angulation of the fragments which were of common occurrence when the old wooden side arm splints were applied previously.

TABLE VII

COMPOUND FRACTURES TREATED

12 cases treated	No deaths	1 infection (pyo.).
11 in males	11 in left arm	Average age, 30 years.

Results

75% had good anatomic results.
78% had good functional results.

Nine additional cases were transferred to the Psychopathic Ward at Bellevue Hospital or died on admission from internal or other causes.

(B) Of the 12 cases of dislocation of the head of the humerus 11 were in the left arm. Four of these cases had an associated fracture of the surgical neck and necessitated open operations for the removal of the head of the bone and insertion of the shaft in the glenoid fossa but always with the same resultant, relatively unsatisfactory functional result. The remaining eight of these cases had fractures of the greater tuberosity and traction in a Thomas splint without any previous manipulation cured both the dislocation and the fracture.

(C) The 29 intra-articular fractures all necessitated careful observation and the institution of movement at an early date so as to prevent stiff joints which would hinder a good functional result.

(D) The seven pathologic fractures were due to chronic cystic degeneration of the bone in five cases, a metastatic carcinoma in one instance and in another a chronic osteomyelitis. Five of these seven cases were in the left arm.

(E) Of the 16 nerve injuries, 13 were paralyses of the radial nerve. One case was a paralysis of the median nerve and the two remaining cases were traumatized ulna nerves. The function, both motor and sensory, returned in every nerve injury case which was observed in the follow up clinic, without

being operated upon. Patience, careful observation and physiotherapy is needed during the six to 12 months which it takes these paralyzed nerves to regenerate and regain their lost function.

(F) Two patients had both the right and left humerus fractured, which naturally greatly complicated their treatment.

(G) The indirect complications were manifold and varied from 171 fractures of other bones in other limbs to 53 internal injuries and medical diseases. They totaled 224 in number, and greatly retarded the patient's eventual recovery and in most instances prolonged somewhat the hospitalization time of the patient. Fractures of the ribs, clavicles and bones of the forearm were the most frequent additional injuries. Thirty-six bones were broken in the lower limbs and exactly half of these were on the same side of the body as the fractured humerus. Skull fractures were relatively rare. Forty-seven cases had both direct and indirect complications associated with the fracture of the humerus. At Harlem Hospital syphilis is not considered a complicating factor in the healing of fractures.

Hospitalization Time.—The length of the hospitalization time varies considerably with the site of fracture and whether or not there were any complicating factors associated with the treatment, such as fractures of other long bones, which by their very nature take longer to heal solidly than do fractures of the humerus. In most instances complications more than doubled the hospitalization time for a patient. For example, in 59 fractures of the upper end of the humerus, 28 were in uncomplicated cases who had a hospitalization period of 16 days, while in 31 complicated cases of similar nature the stay in the hospital was prolonged to 38 days. Then, too, in 56 fractures of the shaft, 25 uncomplicated cases remained only 30 days while the complicated cases stayed for 53 days. The difference is even greater when comparisons are made in 27 cases of the lower end of the humerus where 20 uncomplicated cases were hospitalized for an average period of six and two-thirds days, but the seven complicated cases averaged 27 days each, which is nearly four times as great a length of time. The seven pathologic fractures all had relatively short stays in the hospital, averaging only 20½ days. This is due in a great measure to the fact that it was necessary to immobilize these cases completely in plaster spicas, after first accomplishing the reduction and early union by means of traction in a Thomas splint. These patients were usually discharged to the clinic in their plaster spicas, as firm union is usually delayed because of the intrinsic bone disease.

The 12 compound fractures were divided into seven uncomplicated cases, with an average hospitalization time of 34 days, and five complicated cases whose recovery in the hospital was prolonged to 65 days.

Hospitalization Time Influenced by Type of Traction Used in Shaft Fractures.—The length of the hospitalization time in fractures of the shaft of the humerus also varied greatly as did the final results with the type of traction which was used in treating the different cases. Although the number of shaft fractures is not sufficiently large to draw any definite conclusions,

TABLE VIII

AVERAGE HOSPITALIZATION TIME INFLUENCED BY COMPLICATIONS

142 simple fractures	59 upper extremity	28 uncomplicated cases	2 wks. (16 days).
		31 complicated cases	5½ wks. (38½ days).
	56 shaft	25 uncomplicated cases	4 wks. (30 days).
		31 complicated cases	7½ wks. (53 days).
	27 lower extremity	20 uncomplicated cases	1 wk. (6⅔ days).
		7 complicated cases	4 wks. (27 days).
12 compound fractures		7 uncomplicated cases	5 wks. (34 days).
		5 complicated cases	9 wks. (65 days).
7 pathologic fractures		7 uncomplicated cases	3 wks. (20½ days).
		0 complicated cases	

our results are quite significant and are indicative of the problem confronting hospitals which have an extremely active traumatic service. The average hospitalization time for this type of fracture was lowest when the fracture was treated by "Spanish Windlass Traction in a Thomas Arm Splint," in spite of the fact that this method of treatment was used on those patients who are most difficult to treat and in whom it might reasonably be expected that the results would not be so favorable, nor more quickly obtainable; namely, in the elderly, children, the psychopathic, and patients with compound fractures. The average time for such fractures was 38½ days; while with (Blake's) "Balanced Traction," used only in cases giving a most favorable prognosis, the average time was 41½ days. Only one of the 39 cases treated in "Thomas Splint Traction" was discharged from the wards in a secondary plaster splint because of insecure union, while six of the 15 cases treated in "Balanced Traction" had to be discharged with the secondary plaster splints, this in spite of a more favorable prognosis and a longer hospitalization time. A third method of treatment, "Skeletal Traction," was used in three cases but was quickly abandoned as the results were unsatisfactory, and an added danger of compounding the fracture by insertion of the Kirschner wire, through either the condyles of the humerus or the upper end of the ulna, was deemed an unnecessary procedure which could be dispensed with in favor of the more successful, the simpler, and far less dangerous "Thomas Arm Splint Traction." Add to these already convincing facts that cases treated by "Balanced Traction" gave a very poor percentage of satisfactory anatomic results, as six had unsatisfactory results and only nine of the 15 cases had good results, giving a low rate of 60 per cent satisfactory anatomic results, while the functional results were also somewhat lower than the average. Of the three cases treated with "Skeletal Traction," two had the Kirschner wire inserted through the upper third of the ulna and these both had unsatisfactory functional results and one of these had a poor anatomic

result. The one case in which the Kirschner wire was inserted through the condyles of the humerus had a good anatomic and functional result.

TABLE IX

TIME FOR TYPE OF TRACTION IN SHAFT FRACTURES

39 cases treated in "Thomas Arm Splint Windlass Traction"
Average time in hospital—38½ days.
Used in all types of cases of all ages.
Only one secondary plaster splint upon discharge.
Patient is ambulatory; gives the best results, easiest to apply, easiest to supervise.

15 cases treated in (Blake's) "Balanced Traction"
Average time in hospital—42½ days.
Six secondary plaster splints upon discharge.
Only 60 per cent good anatomic results (lower than the average).

3 cases treated in "Skeletal Traction"
Average time in hospital—39 days.
Two cases had wire through the ulna; poor results.
One case had wire through the condyle; good result.

Bony Union.—The length of time necessary to obtain firm bony union is dependent upon: (1) The age of the patient. (2) The type of fracture. (3) The part of the bone fractured. To these might be added the further factors of: (4) The cooperation of the patient. (5) The skill of the surgeon in manipulating the bony fragments into a satisfactory position so that union may progress more favorably.

Union is somewhat delayed in the elderly, and is usually accelerated in children and robust men of working age. It occurs more rapidly as a rule in comminuted fractures where more blood channels are opened up and an increased amount of free calcium is deposited locally about the area in which the callus is to form. Union apparently is most quickly obtained in supracondylar fractures, partly because of the ample blood supply to this part of the bone and partly because this type of fracture is most frequently seen in children.

Ninety-three of our 167 cases were discharged to the clinic in splints, within two weeks from the day of admission, because their particular fracture no longer needed hospital care but could be ably treated and followed up in the clinic; 18 cases had inconclusive records as to this condition; two cases both with surgical neck fractures were discharged after long confinement, with apparent nonunion, but one of these cases was observed three months later in a follow up, and at that time she had fair union and good function of her arm. This leaves only one known case in which union had not taken place when last observed, and this patient had a follow up of only two months from the date of injury.

Open Operations.—Open operations for the correction of fracture deformities gave very favorable functional results. The unsatisfactory results were due to a certain extent to the nature of the injuries, for of the nine

TABLE X

BONY UNION

93 cases discharged from wards with bony union.
42 cases discharged within 2 weeks to the clinic.
18 cases had records which were inconclusive as to union.
1 case (surgical neck) had delayed union (3 months later had good function—fibrous union).
1 case (surgical neck) had no union (when last observed by us 2 months after injury).

cases operated upon, four were cases in which the surgical neck was fractured and the head of the bone was dislocated. In this type of injury all forms of manipulation and traction are useless and it is often necessary to operate to remove the head of the bone and to insert the upper end in the glenoid fossa. Although these four operations were performed without mishap and the best anatomic results possible obtained, the functional results were not very gratifying. They would probably have been worse if operation had not been resorted to. The remaining five cases which were operated upon all gave satisfactory anatomic and functional results and were performed only when closed reduction was deemed unwise or had been unsuccessful. With the exception of these four cases where the useless head was excised and one case of a badly comminuted compound fracture, which necessitated the removal of a fragment of bone, only four open operations were performed in 162 cases (2½ per cent).

Anatomic Results.—The anatomic results in 150 of the 167 cases were satisfactory and give an average of 90 per cent (Table XI). The best results were obtained in 34 fractures of the lower end of the bone where 97 per cent successful results were had; next come the 56 fractures of the upper end of the bone where good position was obtained in 90 per cent of the cases, and last were the 60 fractures of the shaft of the bone which were manipulated into a correct position and united in 85½ per cent of this type of case. No doubt this percentage would have been even higher if the only form of traction used had been a "Thomas Arm Splint Traction." Seven cases had some bony deformity, six of these had only slight deformity and one a moderate degree of displacement and angulation. Of the 17 unsatisfactory results, 13 were classified as fair and four as poor. Nine of the fair results were in fractures of the shaft, two were in the surgical neck and one was supracondylar. Six of the nine unsatisfactory shaft results were treated in "Balanced Traction," leaving only three of such cases which were treated by the often used "Thomas Splint Traction," and of these three one was a pathologic fracture (osteofibrosis cystica), in which the cystic degenerative changes in the bone became worse, one was a compound fracture of the upper shaft which resulted in a 2 cm. shortening of the bone but a good functional result, while the third case was that of a patient 61 years old who had a Colles fracture of the same limb but who also had a good functional result. The remaining four fair results were in cases already described

where there was a dislocation of the head associated with a fracture of the surgical neck and for which open operations were performed to remove the useless and displaced head of the bone and to place the upper end of the shaft in the glenoid fossa. Three of the four poor anatomic results were fractures of the surgical neck with marked displacement of the shaft, and the fourth was a compound supracondylar fracture which developed osteomyelitis and necessitated removal of part of the lower end of the bone.

TABLE XI

ANATOMIC RESULTS

90 per cent (150 cases) had satisfactory results

150 satisfactory; 90 per cent	83 excellent results.	56 upper extremity, 90	per cent satisfactory.
		60 shaft extremity, 85¼	per cent satisfactory.
	67 good results.	97 lower extremity, 97	per cent satisfactory.
17 unsatisfactory	13 fair results.	6 upper extremity, 10	per cent unsatisfactory.
		10 shaft extremity, 14½	per cent unsatisfactory.
	4 poor results.	1 lower extremity, 3	per cent unsatisfactory.

One case had no union when last observed.
One case had delayed union but good function with fibrous union.
Seven cases some bony deformity (usually slight).
Sixteen cases had nerve injuries (none operated upon).

Functional Results.—The functional results based on a follow up of 106 of the 167 patients whose anatomic results have been cited above or (63½ per cent follow up) gave a slightly less satisfactory percentage of good results than did the anatomic results. However, the percentage of 88 7/10 is gratifying. Of the 94 satisfactory functional results the best percentage was had in the 41 fractures of the shaft of the bone where 92½ per cent good results were recorded. It is worthy of note that it was these same fractures of the shaft which gave the most unfavorable anatomic results when compared to fractures elsewhere in the bone. Fractures of the lower extremity are rated next in the functional results as in 25 cases good results were obtained in 88 per cent. The least satisfactory functional results were in the 40 fractures of the upper extremity of the bone which attained a rating of only 85 per cent. All adult cases with unsatisfactory results had the injury in the left arm (11 of the 12 cases). The remaining case was a supracondylar fracture in the right arm of a child who did not continue with his physiotherapy treatment in the clinic after discharge.

Of the 12 unsatisfactory functional results nine were classified as fair and three as poor. The three poor results were all in fractures of the surgical neck, but the nine fair results were equally divided with three each in the upper end, the shaft, and the lower end of the bone. Therefore, six of the 12 unsatisfactory functional results were in fractures of the surgical neck of the bone, but of these six cases four were cases which had a concomitant dislocation of the head of the bone and another was the one case of nonunion already mentioned. In fractures of the shaft better functional

results were observed in those cases treated in "Thomas Arm Splint" with "Spanish Windlass Traction" than in those cases treated in either "Balanced Traction" or " Skeletal Traction." Two of these midshaft fractures (one treated in Thomas splint traction and one in "Balanced Traction") had unsatisfactory anatomic results. In the two cases treated by a Kirschner wire through the ulna (for skeletal traction), the functional results were both poor, while in the one case with the wire inserted through the humeral condyles the function was satisfactory. The average hospitalization time for the patients with unsatisfactory functional results was 47 days, for satisfactory results 35 days.

Although there were 85 direct complications, consisting of nerve injuries, cystic degenerations of the bone, compounding wounds, dislocations and intra-articular fractures, the satisfactory functional results were 89 per cent. Of the unsatisfactory functional results five were in cases who had open operations and two were in cases of skeletal traction through the ulna. In addition there were 171 other bones fractured in the body and there were 53 cerebral or internal complications of serious importance, all of which had to be treated at the same time as the fracture of the humerus. It is only natural that the functional results under discussion were somewhat impaired in the effort to save the patient's life or prevent any serious deformity as a result of his many injuries (Table XII).

TABLE XII

FUNCTIONAL RESULTS

89 per cent (90 of 106 cases) had satisfactory results based on a follow up of 106 cases.

94 satisfactory; 89 per cent	34 upper extremity, 85	per cent satisfactory results.
	38 shaft extremity, 92½	per cent satisfactory results.
	22 lower extremity, 88	per cent satisfactory results.
12 unsatisfactory; 11 per cent	6 upper extremity, 15 (3 fair—3 poor).	per cent unsatisfactory results
	3 shaft, 7½ (fair).	per cent unsatisfactory results
	3 lower extremity, 12 (fair).	per cent unsatisfactory results

Six of the twelve unsatisfactory results were in the surgical neck.
The only three poor results were in the surgical neck.
Four unsatisfactory results were after open operation.
Two unsatisfactory results were after skeletal traction.
Eleven of the twelve cases were in the left arm.

Treatment.—The type of treatment which should be used varies greatly with the regimen of the particular hospital, and the amount of supervision each case can be given, and also with the location of the fracture. Fractures in the surgical neck are best treated in "Thomas Splint Traction," but when associated with a dislocation of the head of the bone an open operation may have to be resorted to, so that the displaced and useless head may be removed and the upper end of the shaft inserted in the glenoid fossa.

This type of case and compound fractures are in our opinion the only indications for operation at an early date.

Impacted fractures of the surgical neck with the fragments in good position can usually be treated satisfactorily in a Velpeau bandage without any previous traction.

Fractures of the greater tuberosity likewise are usually easily treated in a Velpeau bandage, but if displacement of the fragments or a dislocation of the head of the bone exists, traction in a Thomas splint will be necessary, and this form of treatment has never failed in reducing both the fracture and the dislocation.

Fractures of the shaft, as has already been shown, are best treated in hospitals which have a very active traumatic service, by means of "Spanish Windlass Traction in a Thomas Arm Splint" applied over a plaster buttress (Fig. 1), which will protect the axillary structures from the trauma of the

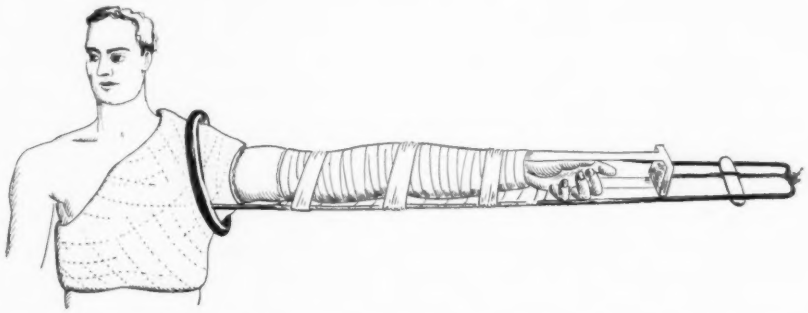

FIG. 1.—Illustrates the method of employment of the "Spanish Windlass Traction in a Thomas Arm Splint."

ring of the splint and will diffuse the resultant pressure of the traction away from the axilla and over the whole lateral chest wall. This method of treatment makes it possible to get the patient out of bed within two weeks without any danger of disturbing the position of the fragments. It is easily applied and adjusted and greatly simplifies supervision by the ward nurses and the doctors; in addition it immobilizes the fragments better than any other form of traction and gives firmer union more quickly and has terminated with better anatomic and functional results in our experience. Open operation for a shaft fracture should rarely, if ever, be necessary, and in this series there was only one such case in which control of the fragments was lost while endeavoring to apply a plaster splint at too early a date, after having obtained an excellent anatomic position by "Thomas Splint Traction." If there should be a lateral displacement of the fragments which was not corrected by this traction, then two small slings or cuffs passed about the arm above and below the fracture site and tightened to the opposing side bars of the splint will usually remedy the situation.

Supracondylar fractures should have an immediate reduction of the fragments under anesthesia. Reduction is best accomplished by applying

steady manual traction and then manipulating the fragments into place and acutely flexing the forearm on the arm in a Jones position to lock the fragments in place by the action of the triceps muscle. If manipulation proves unsuccessful, traction in a Thomas splint gives a good result, but increases the hospitalization time. Motion should be begun in these cases at a very early date, just as it is in intra-articular fractures.

Compound fractures necessitate an immediate débridement of the wound and either a packing of the wound with gauze, if a loss of skin substance exists, or immediate suturing. The fracture is then treated as any simple fracture of the same type.

In cases where an associated nerve injury exists, the fracture should be treated as though it were uncomplicated. If it be definitely known that the nerve has been completely severed and not merely crushed, then it will be necessary to suture the nerve as soon as all danger of compounding the fracture has passed. However, in this series of cases no such condition presented itself. In 16 cases of nerve injuries, suturing was never necessary and function returned to all cases who were observed in our follow up clinic. If there is any doubt as to whether or not the nerve is severed it is best to err on the side of being conservative and not operate for at least six months and then only when no sign of regeneration has occurred. If the radial nerve is the injured member it is best to apply a "cock-up" splint to support the extensor muscles and prevent them becoming over stretched while awaiting the return of the nerve function. All muscles supplied by a nerve which has been traumatized should receive frequent physiotherapy.

CONCLUSIONS

(1) The site of the fracture tends to vary with the age of the patient, for the older the patient the higher the level of fracture and the younger the patient the lower is the level of the fracture. The midshaft was the most frequent site for patients within the working age group.

(2) (*a*) Sixty-one per cent of the fractures of the humerus were in the left arm.

(*b*) Eleven of the 12 compound fractures were in the left arm.

(*c*) Eleven of the 12 dislocations of the head of the humerus were in the left arm.

(*d*) Eleven of the 12 unsatisfactory functional results were in the left arm.

(*e*) Five of the seven pathologic fractures were in the left arm.

(3) A buttress spica will protect the axillary nerve structures and also make the patient more comfortable as it diffuses the pressure of the ring of the splint over the whole lateral chest wall, and it will also permit making the patient ambulatory at an earlier date.

(4) Do not use skeletal traction through the ulna as traction on the forearm with the elbow flexed apparently strains the ligaments and impairs function of the elbow joint.

(5) Early reduction under anesthesia is the treatment of choice for supracondylar fractures. Immobilize in Jones position and start physiotherapy and motion at an early date to obtain good functional results.

(6) Open operations should rarely be necessary to obtain good results.

(7) In compound fractures do an immediate débridement and then treat your fracture as if it had not been compounded. The results will be excellent if conservatism is practiced (11 of the 12 cases of this series healed cleanly).

(8) Institute physiotherapy treatment early. It stimulates callus formation while the arm is in traction and facilitates function later. This is especially so now since diathermy has become so successful and to a great extent has replaced baking and massage.

(9) In our experience the "Thomas Splint Traction" gives better union more quickly and necessitates a shorter hospitalization period. It can be used in all types of fractures both as an emergency measure and as a method of treatment and in all types of patients of all ages. Its use resulted in the best anatomic and functional results. In hospitals where a less active service is found, and in those whose endowments permit more nursing supervision, it is quite likely that the treatment of choice in many instances would be the "Balanced Traction."

(10) The predominating factors in our unsatisfactory results, both anatomic and functional, were (*a*) the presence of a dislocation of the head of the bone associated with a fracture of the surgical neck; (*b*) the lack of cooperation by patients who were either chronic alcoholics or mental cases; (*c*) the multiplicity of the various complications, both direct and indirect; (*d*) the institution of skeletal traction through the ulna with the elbow flexed; and (*e*) the severity of comminution or compounding of the fracture itself.

(11) Our best anatomic results were obtained in supracondylar fractures and the worst in fractures of the shaft. The best functional results were obtained in shaft fractures and the worst in fractures of the upper extremity of the bone. Fractures of the shaft, therefore, gave the lowest percentage of satisfactory anatomic results but the highest percentage of functional results.

(12) Finally with these simple and inexpensive methods of treatment we obtained 90 per cent satisfactory anatomic results; 89 per cent satisfactory functional results; and had only one case of nonunion.

OSTEITIS PUBIS FOLLOWING SUPRAPUBIC PROSTATECTOMY

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A SURVEY of the literature reveals an astonishing paucity of cases of osteitis of the pubis following suprapubic prostatectomy. It is even more surprising to note how little orthopedists and interns know about this clinical entity. It may be that this condition is rare, but it seems more likely that some, due to their mild character, are completely overlooked, and the others incorrectly diagnosed.

Thirteen years ago I noted within an interval of a few months two cases of severe pain over the symphysis pubis with adductor spasm of the thighs. Both of these cases were in men in the late sixties, following a two stage suprapubic prostatectomy. The complication occurred a few weeks following their discharge from the hospital. Having had no experience with this condition, it was believed that we were dealing with metastases involving the symphysis pubis from glands which, though clinically benign, might have actually been malignant. In one of the cases, due to the severity of the symptoms and signs, it was believed that we were dealing with an osteomyelitis of the pubis, and an exploratory operation was performed, but nothing of note found. With the view that these cases were hopeless, both were placed upon sedatives, and after a prolonged period of confinement to bed, the symptoms and signs completely disappeared.

Aschner states that this complication was also encountered after suprapubic operations upon the bladder other than prostatectomies. Beer[1] first encountered it in 1916. The only mention made of this clinical entity in textbooks on urology is by H. Cabot.[2] It appears that the complication only follows suprapubic operations in which the bladder is opened. It is difficult to state the exact cause of the inflammation of the periosteum and bone. Beer mentions the possibility of compression injury of the pubis by retractors in the course of the operative procedure, and also pressure due to the suprapubic drainage tube. These seem very unlikely since in the case herein described the operation was extremely simple, requiring but very little retraction, and the drainage tube was not large and made of soft rubber.

Symptoms.—Symptoms usually arise from two to three weeks after the enucleation, beginning with pain in the lower angle of the wound which grows severe when the patient attempts to sit up and when coughing or sneezing. This is due to pulling of the recti abdominis muscles against the inflamed periosteum of the symphysis to which they are attached. As the disease progresses the pain involves the body and descending rami of the pubis and the adductor muscles attached to this part of the pelvis, so that walking and separation of the thighs becomes extremely painful. Patients

have a peculiar wabbling gait. Each step is taken deliberately with the aid of a cane or crutch, since the slightest jarring greatly aggravates the pain. The patient finds it extremely difficult to move about in bed, and also experi-

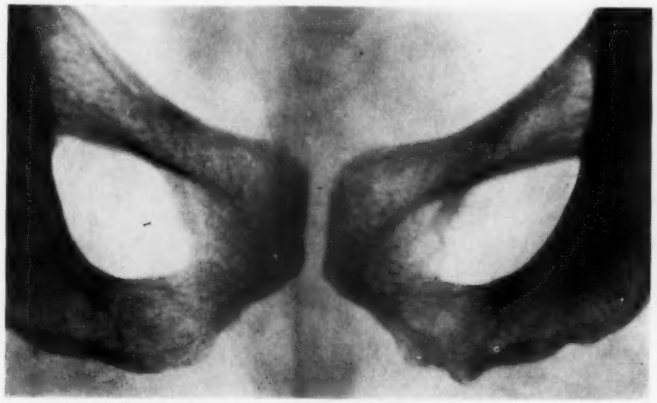

FIG. 1.—Radiogram of the pelvis taken October 24, 1933, the onset of the illness, showing beginning erosion of the upper margin of the symphysis pubis.

ences intense pain in the thighs and lower abdomen during defecation or at the end of micturition.

The temperature may show a slight to a moderate elevation. On examination one usually elicits exquisite tenderness over the symphysis pubis, over

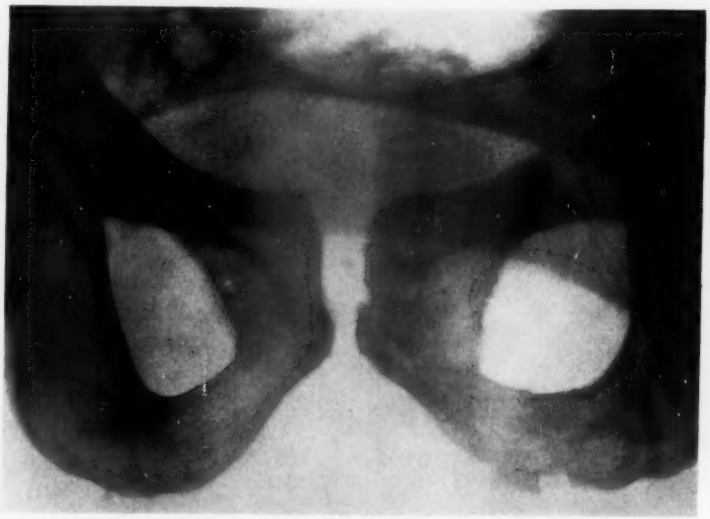

FIG. 2.—Radiogram of the pelvis taken November 25, 1933, showing a more extensive erosion of the symphysis pubis.

the lower attachment of the recti abdominis muscles, over the body of the descending rami of the pubis, and over the adductor muscles of the thighs. As the process advances, the temperature drops to normal but the signs and symptoms progress.

A roentgenogram taken in the early stages of the disease may show nothing at all, or only a slight fraying of the superior corners of the symphysis.

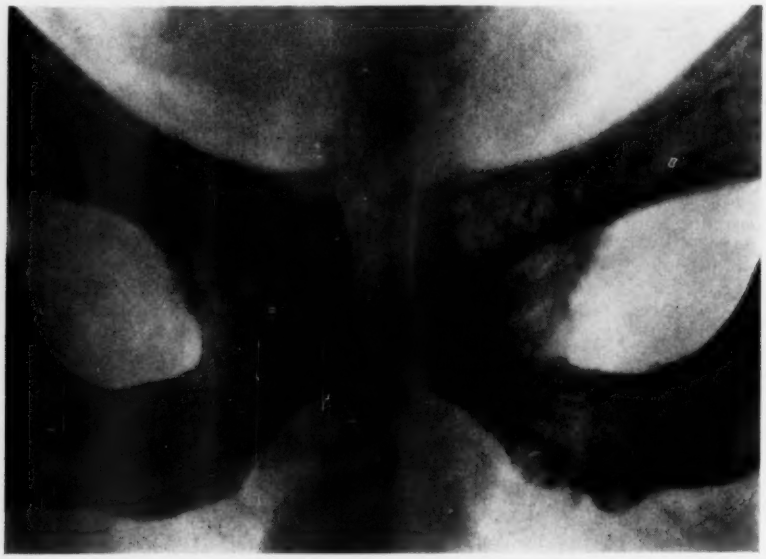

FIG. 3.—Radiogram of the pelvis taken December 8, 1933, showing still further destruction of the symphysis pubis with a tendency toward a V shaped defect in the symphysis.

Later one notices a definite rarefaction of the body and descending ramus of the pubis, and it is in this stage that the unwary will diagnose the process,

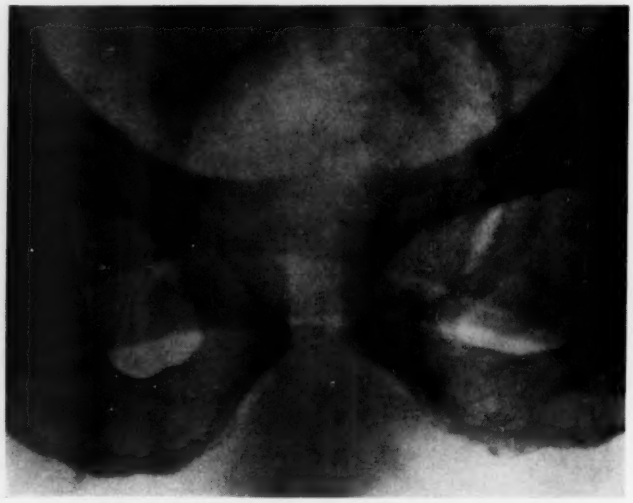

FIG. 4.—Radiogram of the pelvis taken December 23, 1933, showing a leveling out of the destructive process of the symphysis, giving the appearance of a separation of this portion of the pelvis.

especially when the temperature is elevated, as an osteomyelitis and advise surgical intervention; or when the temperature is normal the osteoporosis

will be looked upon as due to metastatic deposits. The next stage is an absorption of the symphysis beginning at the superior border and advancing caudad to the inferior pubic arch, so that a V shaped defect can be seen. Later, as the absorption continues, the picture is that of a separation of the symphysis. Although in a few of the reported cases the process has gone on to bone necrosis with formation of a sequestrum, this is the exception rather than the rule.

The disease must be differentiated from osteomyelitis and from metastatic involvement of the pubis. To one who has never been confronted with this clinical entity, the differential diagnosis is difficult. In the early stages, the symptoms and signs, coupled with positive roentgenographic evidence of bone destruction and fever, make the diagnosis of osteomyelitis very suggestive. In the later stages, in the absence of fever, but with the roentgenographic evidence of extensive osteoporosis, one is strongly led to suspect metastatic deposits. To one who has had experience with this complication the diagnosis of osteitis pubis stands until disproved. Should the process prove to be due to osteomyelitis, the temperature will remain elevated or rise, and a sequestrum will be thrown off and an abscess eventually form. In the event the lesion is due to metastases, other symptoms will become manifest, such as cachexia and loss of weight. The roentgenogram will also disclose areas of osteosclerosis along with osteoporosis, particularly around the sacroiliac joints.

As regards treatment, it must be understood that the disease is a self-limited one, and there is no specific curative agent. The most important feature in such a patient is to see that no one attempts operative intervention, except in cases where as abscess develops which requires evacuation, and these are extremely rare. Since there are no specific measures available, the treatment consists of such palliative measures as rest in bed, baking, massage, and hot sitz baths. In the severe cases the best treatment is the application of a plaster case reaching from the midabdomen over both legs as far as the ankles. The diet should be rich in vitamins and should contain an abundance of calcium. Symptoms may persist from two to six months and then recede.

Case Report

N. E. B., a physician, aged 56, was first seen August 23, 1933, complaining of difficulty in starting urination and nocturia, over a period of several years. Cysto-urethroscopic examination revealed a marked enlargement of the middle and lateral lobes of the prostate. Residual urine one and one-half ounces. Following the examination the patient had chills and fever of 105° lasting 48 hours, following which the temperature receded to normal. Physical examination was essentially negative save for an enlarged, elastic prostate gland felt per rectum.

August 28, 1933, under spinal anesthesia, a suprapubic cystotomy was performed and a medium sized soft rubber drainage tube introduced into the bladder. Following the operation the patient did very well, and September 13, under spinal anesthesia, the prostate was enucleated in one piece without difficulty. Two small packings were left in the prostatic bed which were removed after 48 hours. The patient passed through a smooth, uneventful, afebrile convalescence and began to void on the seventh day, and

was discharged from the hospital September 26. The pathologic diagnosis of the specimen was glandular (cystic) hypertrophy of the prostate.

About one week after leaving the hospital, which was about three weeks after the prostatic enucleation, he began to experience pain in the left suprapubic region intensified by movement, coughing and walking. He also noted a dull pulling sensation in both lower extremities, particularly the left, and over the inner aspects of the thighs and calves. The pain was just as severe at night as during the day, and was aggravated by the slightest motion. At the end of urination he experienced spasmodic contractions of the adductor muscles of the thighs with pain reaching down to the calves. The pain was more severe on the left side.

The physical findings October 30 revealed the patient walking with the aid of a cane with great difficulty, the gait being wabbly. Difficulty was also noted in sitting and arising. There was a 60 degree flexion contracture of the left hip with marked spasm of the adductor muscles. There was a slight atrophy of the left thigh. Palpation of the pubis revealed exquisite tenderness over the symphysis, the lower ends of the rectus muscles and the left ramus of the pubic. There was no evidence of abscess formation, nor was there any limitation of motion of the right hip. The temperature was 99.4°. A roentgenogram (Fig. 1) disclosed a haziness and beginning bone destruction of the left pubis.

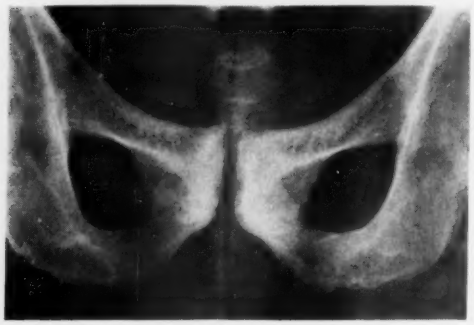

FIG. 5.—Radiogram of the pelvis taken November 24, 1934, showing almost complete restitution.

For about two weeks the symptoms became much more severe, so that he found it increasingly difficult to get about. The temperature reached as high as 101°, following which it became normal for the remainder of the disease. The pain gradually spread to the left ramus and adductor muscles and over the attachment of the recti muscles to the symphysis. There was no indication of abscess formation during this period.

Hot baths, baking and massage failed to give relief. A plaster case reaching from the umbilicus over both thighs to just above the ankles was applied November 9. After one week the case was removed and he was given short wave length diathermy, but without appreciable relief. A second case was applied November 16, but owing to the great discomfort which it caused, it was removed one week later. Orthopedic consultants advised wiring of the symphysis because of the increasing separation; others incision and drainage on the assumption that it was an active osteomyelitis. Radiograms (Figs. 2, 3 and 4) taken at intervals revealed a progressive destruction of the symphysis.

A third plaster case was applied December 11, and left on for seven weeks, following which the symptoms rapidly disappeared. A communication on May 10 stated that he had discarded his cane and was feeling well, except for slight pain in the thighs when spreading his legs, fatigue when walking fast or exercising, and a sense of discomfort over the sacroiliac joints (Fig. 5). March 6, 1935, the patient reported that he had no further symptoms.

SUMMARY AND CONCLUSIONS

(1) Osteitis pubis is a rare complication following surgical procedures carried out on the bladder through a suprapubic incision.

(2) The etiology of the disease is unknown.

(3) The outstanding features of this complication are pain localized over the lower attachments of the recti abdominis muscles, the body, symphysis and rami of the pubic bone, the inner aspects of the thighs with adductor spasm, so that the patient finds it painful to walk, to spread the thighs, and to cough. A late symptom is pain on defecation and terminal dysuria.

(4) Fever is rarely present throughout the disease, but may be present during the early stages.

(5) Symptoms usually occur about two to three weeks after operation.

(6) The process very rarely goes on to suppuration and fistulous formation.

(7) Since the disease is a self-limited one, the treatment is palliative except in the few cases where suppuration occurs. Most of the cases will respond to baking and massage and to the application of a properly fitted plaster case.

(8) The disease is self-limited with symptoms lasting from two to six months, followed by resolution and complete recovery.

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BOOK REVIEWS

DISEASES OF THE LIVER, GALLBLADDER, DUCTS AND PANCREAS. THEIR DIAGNOSIS AND TREATMENT. By Samuel Weiss, M.D. Quarto of 1099 pages, illustrated. New York, Paul B. Hoeber, Inc., 1935.

The author's "main intention has been to place before the student and general practitioner facts which will aid them in their daily contact with patients." It consists of 31 chapters and 358 illustrations and is encyclopedic in its range, bringing the literature on the liver, gallbladder, *etc.*, up to June, 1935. As a reference book it has great possibilities for usefulness. To be used for a continuous system of teaching it is overamplified and includes a surprising amount of distinctly laboratory material and technical procedure. The style adopted by the author is terse and reliance is placed to an unusual degree upon the short paragraph for the presentation of isolated clinical facts. It is occasionally very difficult to follow the relationship of a given statement with what precedes it and with what follows it—for example, on p. 141 appears: "Antitoxic Functions. The liver destroys certain toxic substances entirely, or else combines them with other substances to form non-toxic bodies which are excreted." No less than four pages farther on there appears "detoxifying protective action of the liver" where it is stated impairment of such detoxifying action may contribute to the causation of cholemia. It is interesting to note that apparently Adami's contribution on subinfection was entirely missed in the study of this liver detoxifying activity.

Chapter VI on the Indirect Examination of the Liver is well considered and contains such a wealth of material that it perhaps would be somewhat better if greater selectivity were exercised.

The economic and public health importance of the typhoid carrier would seem to merit a somewhat more extended treatment. The cure of the typhoid carrier by cholecystectomy is only incidentally mentioned. Chapter VIII on Roentgenology of the Liver, Gallbladder and Pancreas is by A. Judson Quimby and is given ample proportions and in consonance with present day teaching.

The reader is surprised that in Chapter IX, Functional Diseases of the Liver, the theory of lithemia of the vintage of 1877 should be given 17 lines and it is not without interest that "biliousness or torpid liver" is raised to the dignity of a concrete disease. On p. 276, under Classification of the Functional Derangements of the Liver, there is listed a congeries of symptoms that is strangely reminiscent of the alleged symptomatology of chronic intestinal stasis. This method is strongly suggestive of the continental system wherein a name is given to a supposed disease entity and then every possible deviation from the normal is listed under it as a symptom.

Chapter XIV—Acute Yellow Atrophy of the Liver—is very well done, except for the tendency to make categorical statements that are not in keeping

with present day thought—"Catarrhal jaundice is an infectious disease; acute yellow atrophy of the liver is an aggravated form of it." Icterus catarrhalis is neither a morbid nor pathologic entity and the icterus that is associated with the condition may be due to degenerations and multiple necrosis of the liver, hematogenous in origin but without known etiologic factors.

Chapter XXII is devoted to Jaundice and is informative but lacks a logical presentation of the essential chemistry of bilirubinemia. A broad review of the chemistry of jaundice would be especially valuable in indicating the diagnostic distinction between so called "painless jaundice" and "painful jaundice." The author has presented his own classification of jaundice based upon the etiologic factors producing it, necessitating over four pages to list all of the possible conditions that might cause jaundice. However, McNee's classification, which is by all odds the simplest and most workable, is covered in seven lines, to be followed by four paragraphs of explanation some two pages farther on.

Courvoisier is listed five times in the index of personal names yet nowhere does there appear a logical explanation of the Courvoisier gallbladder and its importance in diagnosis. Bilirubinemia is quoted once in the index and is in the text under the test for hemoglobin.

Surgery of the Gall Bladder, Bile Ducts and Liver is contributed by Dr. J. Prescott Grant and covers 36 pages and indicates the operative procedures of the contributor's personal technic. What is presented is sound and in consonance with modern surgical opinion, yet the reviewer is conscious of a lack of precise information in dealing with the complications that are encountered in gallbladder surgery and particularly in those patients who were operated upon in the presence of jaundice.

In Chapter XXVIII, devoted to Disease of the Pancreas, there is an unusual tendency to abbreviate which is not apparent in other sections of the book. Out of 40 pages devoted to diseases of the pancreas approximately 13½ pages are given to illustrations, which add little to the text and are not in themselves explanatory. The reader misses Deaver and Pfeiffer's contribution on the association of gallbladder disease and pancreatitis, as well as the contribution of Arthur Cabot on the Courvoisier Gallbladder. There are included, however, such recent contributions as the consideration of hyperinsulinism by Seale Harris and "dinitrophenol jaundice." It is to be regretted that Whipple's masterly paper on adenoma of the pancreas apparently was published too late for abstraction.

In the section on Malignant Tumors of the Pancreas, Courvoisier's name does not appear and what is of more particular interest is that the author fails to make any recommendation for treatment and the palliative operation of cholecystenterostomy is not mentioned. This is in contrast with the last sentence on p. 822, "For surgery of the pancreas the reader is referred to books dealing with the subject." This is the first distinct omission of the author's opinion on surgical indications which up to this point have been unusually sane and liberal.

Chapter XXIX, Methods of Determining Pancreatic Ferments, embraces 23 pages of text that might well have been omitted, for this material is distinctly within the province of text-books on clinical pathology.

Chapter XXX, Dietetic Management of Diseases of the Liver, Gall Bladder and Pancreas, discusses diet and assembles the various regimens that have had varying degrees of usefulness in the treatment of disease of the biliary system.

The few animadversions indicated above in no way detract from what is a splendid presentation of the subject. The collecting and collating of the material represents a stupendous labor. It is essentially an epitome of practically everything written about the subject matter as of June, 1935. The major defect of the book is the over simplification by extensive subdivisions of text. This renders it exceedingly difficult for the reader to preserve any sense of the continuing process of disease and results in a sort of interrupted pattern of diagnostic writing. It is suggested that in any subsequent edition the text be submitted to ruthless pruning, and that it be printed on lighter weight paper—as the handling of a book weighing seven and one-half pounds for any length of time, on anything but a desk, is difficult.

CHARLES GORDON HEYD, M.D.

DISEASES OF THE THYROID GLAND. ARTHUR E. HERTZLER, M.D. C. V. Mosby Co., St. Louis, 3rd edition, 1935.

In these days when most medical articles are little more than a maze of statistics and references, it is refreshing to come across a book which omits percentages and bibliography entirely and is intentionally devoted to the expression of the author's own experiences and impressions. This third edition of Doctor Hertzler's Diseases of the Thyroid Gland is thoroughly his own book, a detailed, lucid presentation of his personal conclusions, based on many years' experience with thyroid patients and close acquaintance with the practice in other leading thyroid clinics. Although the text-book character of previous editions is superficially maintained by the complete, well indexed arrangement of chapters and headings, it reads more like an original thesis. It is really a new book, having been radically revised and rewritten. Because of its personal and authoritative nature it should arouse widespread interest and criticism, and may well have considerable effect on the development of newer trends in the treatment of thyroid disease and lead to better coordination of our present ideas.

The most stimulating part of the book is probably the new introduction, entitled "General Considerations." Here, the author has aptly singled out the problems which most need discussing today—the fact that hyperthyroidism is more than simple hypersecretion of the thyroid, the problem of the interrelation of simple goiters and the toxic and degenerating types, the difficulty of relating anatomic and physiologic changes with clinical findings, and lastly the all important question of what would happen to goiters if we did not

operate upon them. The importance of personal follow up of operated and non-operated cases is, of course, stressed. The questions raised, as well as the occasional difficulty in answering them convincingly, bring forcefully to our attention the need of further research in this field.

Doctor Hertzler's solution of these problems is based on his conception of thyroid disease as a "continuous process." In agreement with this conception he proposes a new classification of thyroid affections, which has many academic merits, but which will be generally criticized as being too unwieldy for clinical use. The rest of the book suffers a little from the necessity of conforming to this classification, particularly in treating as a separate clinical entity, the "degenerated colloid goiter" as distinct from the ordinary adenomatous types of colloid goiter. The author uses as the basis for this clinical diagnosis the well known degenerative and involutionary changes found in the end stages of both toxic and non-toxic goiters, but he admits that both the clinical and pathologic findings in this group are very variable, and we are left with the impression that it is a decidedly confusing and unsatisfactory diagnosis from the point of view of the clinician. The point is well taken, however, that the markedly degenerative type of gland indicates long standing disease with attendant chronic systemic disturbances, frequently cardiac. In both the toxic and the non-toxic cases of this type, subtotal thyroidectomy usually benefits the constitutional symptoms.

The chapters on etiology and on goiter in childhood are particularly well presented. For the young thyroid surgeon, the chapters on morphology and topographic anatomy of the thyroid, as well as the chapters on hospital management of patients (written by Dr. Hertzler's resident surgeon, Victor E. Chesky, M.D.) and on operative technic should be of interest. The illustrations by Tom Jones are, of course, excellent. In Boston we would consider some of Hertzler's operative technic, such as operating without face masks, a little unorthodox, and we do not have a great deal of use for local anesthesia which he recommends exclusively. On the other hand, he is scornful of the unskilled operator who must resort to general anesthesia. The truth of the matter is that equally excellent results are obtained with several different technics and anesthetics. It is primarily the surgeon rather than the technic which is the important variable factor.

Taken all in all, this latest edition is decidedly stimulating and worth while. Any clinician interested in thyroid disease, particularly thyroid surgery, will be well repaid by reading it through and forming his own opinion regarding the problems discussed so thoroughly and competently by the author.

LEWIS S. PILCHER, 2nd, M.D.

MEMOIR

HAYWARD W. CUSHING

1854–1934

Dr. Hayward W. Cushing was born in Boston in 1854, was of Harvard, 1877, was graduated from the Medical School in 1882, studied abroad, served Children's, City and Carney Hospitals, was a member of all the proper things, retired in 1905, died at 80, in 1934.

Cushing's life and death bring back the tragedy of his retirement at 51, under a mistaken diagnosis of malignant disease. Professionally, that was final, though he regained and retained health and vigor and his unusual mentality nearly to the end.

The history of Doctor Cushing lies far back, before 1905, and on my desk lie the titles of publications by him in the 80's and 90's and the record of the "right angle" continuous intestinal suture, the one thing sometimes recalled today. This dates 1899.

Nephrectomy for calculi (successful), 1892; improved method for hernia operation, 1888; excision elbow under cocaine, 1887; hernia under local anesthesia, 1898, and again in 1899; typhoid cholecystitis, 1898; successful operation on wound of thoracic duct, 1898; successful operation on traumatic cyst of pancreas, 1898; jejunal fistula, 1899; splenectomy in splenic anemia, 1899; typhoid perforations, 1898; hematomyelia, 1898.

Before his retirement he knew more, had thought out more things, than the rest. He operated as he thought—slowly, carefully, but his slow operations were so planned, so carefully, so gently done that he got amazing results. His nickname of "Careful Cush" carried recognition of both these items and we held him in enormous regard. He and Elliot, also early retired, were doing intestinal surgery, for instance, in the last century and doing it well when the others could not.

A scholarly medical man, a very competent surgeon, he cared not a whit for applause, was indifferent to private practice, concerned with his professional interest and research alone. An odd type by present day standards, but we, his juniors, respected him, learned much from him, loved him.

Frederic Jay Cotton.

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